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TECHNICAL MANUAL

NOTES ON CARDIOLOGY IN AVIATION MEDICINE

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WAR DEPARTMENT,
WASHINGTON, *November 12, 1940.*

NOTES ON CARDIOLOGY IN AVIATION MEDICINE

Prepared under direction of
The Surgeon General

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CHAPTER 1

GENERAL

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SECTION I

EMBRYOLOGY OF HEART

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1. Derivation.—*a.* The cardiovascular system of the mammal is derived from the mesodermal layer of the embryo. Very early in the differentiation of the cell masses, vascular areas develop which consist of plexuses of angioblastic cells. Both vessels and blood cells are derived from these vasoformative areas. Thus very early in the process of development a vascular net forms with a capacity sufficient to convey the nourishment needed from yolk sac to the rudimentary organs.

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b. The heart itself is derived from the first two of these vessels formed, the primitive aortae. These vessels in their earliest stages run longitudinally, one on each side of the notochord. As the head forms and the embryonic area undergoes cephalic bending, the primitive aortae also bend, and their anterior portions come to lie beneath the foregut as hook-shaped projections. About the thirteenth day the primitive aortae fuse for a short distance beneath the foregut into a structure known as the primitive cardiac tube. At this stage the primitive cardiac tube has two arteries or aortae proceeding from its cephalic portion and two venous trunks emptying into its caudal portion. The aortae, after undergoing several stages of developmental change through a series of aortic arches, become the adult aorta and adult pulmonary artery. The venous trunks are each formed by three veins, the lateral umbilicals, the vitellines, and the anterior cardinals. These, through another intricate series of developmental changes, become the superior and inferior venae cavae.

2. Development.—*a.* As development proceeds the primitive cardiac tube becomes separated by constrictions into five portions, which are from posterior to anterior, sinus venosus, atrium, ventricle, bulbis cordis, and truncus arteriosus. Of the constrictions, the only one of importance is the atrioventricular. From it arise the future auriculoventricular valves and the auriculoventricular node. Also from it arises the septum intermedium discussed in *b* below. Shortly after the constrictions appear the tube begins to bend upon itself to form an S-shaped structure, the anterior portion bending over to the right and the posterior portion bending over to the left. As growth continues and accentuates the bending of the tube upon itself, the sinus venosus and truncus arteriosus become adjacent structures. Thus two limbs of a loop are formed, the caudal of which contains the sinus venosus, the atrium, and a portion of the ventricle and the cephalic of which contains the truncus arteriosus, bulbis cordis, and the remaining portion of the ventricle. In this manner is formed the forerunner of the base and apex of the adult heart, the veins and arteries at the base and the ventricle at the apex.

b. About the time the cardiac tube is completing the bending process, other developments are taking place, namely certain fusions and absorptions plus the division of the atrium, ventricle, and truncus arteriosus. The sinus venosus is absorbed into the atrium, the right horn of the sinus becoming the sino-auricular node and the left horn becoming the coronary sinus. The bulbis cordis is absorbed into both the truncus arteriosus and the ventricle. The part absorbed into the truncus arteriosus merely lengthens that section, while the portion ab-

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sorbed into the ventricle becomes the infundibulum of the right ventricle of the adult heart. The atrium divides into right and left auricles by the growth of three septa, septum primum, septum secundum, and septum intermedium. The septum primum and secundum grow from the superior portion of the atrium, whereas the septum intermedium grows in from the anterior and posterior portions of the atrial wall near the atrioventricular constriction. The septum secundum and intermedium unite to form the interauricular wall, leaving in their fusion an opening called the foramen ovale. The septum primum grows over the foramen ovale on the left side of the fused wall, acting during foetal life as a flap valve, preventing backflow of blood from the left to right auricles. After birth it fuses with the septum, closing off the foramen ovale to form the fossa ovale. The ventricle divides by growth of a muscular septum from the lower portion of that chamber. This unites with the lower portion of the septum intermedium to form the interventricular septum, the superior section of which is membranomuscular in the adult heart and the inferior section entirely muscular. The truncus arteriosus divides into the basal portion of both the aorta and pulmonary artery by an intricate process of septal growth which ultimately brings the aorta and pulmonary artery into proper relationship with their respective ventricles. The semilunar valves arise from infoldings in the wall of the truncus arteriosus at its proximal portion. The heart has now accomplished its primary metamorphosis and has acquired the same form and structural characteristics as the adult heart.

3. Properties of foetal heart.—It should be noted in passing that all during the phases of development from primitive cardiac tube to adult heart, the structures have been performing their function of propelling blood through the cardiovascular system. The cardiac tube from its inception has possessed all of the fundamental properties of adult heart muscle. The power to initiate stimuli is especially well developed in the sinus venosus and, as a result of this, the contractions of the embryonic tube start in the sinus venosus. This function is retained throughout life by this particular area as it becomes the adult sino-auricular node. Thus even before the heart chambers are differentiated, the primitive cardiac tube undergoes rhythmic contractions and assumes the function of a rudimentary heart.

4. Foetal circulation.—At the time of birth the two auricles are in direct communication with each other by means of the foramen ovale. This opening in the interauricular septum permits the flow of blood from the right auricle into the left auricle without the neces-

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sity of passing through the right ventricle. A certain amount of blood does pass into the right ventricle, however, and from there through the pulmonary artery. Very little of this blood passes through the lungs as the major portion is deflected through the ductus arteriosus into the aorta. The ductus arteriosus is a vestige of the complex multiple aortic arch fusion and absorption mentioned in paragraph 1*b*. Thus the flow of blood through the foetal heart begins with the inflow of blood from the venae cavae into the right auricle where it becomes divided into two routes. The major portion passes through the foramen ovale into the left auricle, to the left ventricle, and thence to the systemic and placental circulations. The minor portion passes through the right atrioventricular opening into the right ventricle, through the pulmonary arteries and ductus arteriosus into the aorta and general circulation. Foetal blood passing through the heart is essentially an admixture of venous and arterial components due to the confluence of the pulmonary and systemic circulations and the fact that all oxygen and nourishment is obtained from the placentra. At birth the functional necessity for the foramen ovale and ductus arteriosus ceases with the aeration of the lungs, and shortly thereafter the former closes by fusion of the septum primum and the latter atrophies into a vestigial cord.

5. Recommended texts.

Textbook of Anatomy (Cunningham)	William Wood and Co.
Gray's Anatomy (Lewis)	Lea and Febiger.
Textbook of Embryology (Bailey-Miller)	William Wood and Co.
Developmental Anatomy (Arey)	Saunders.

Section II

ANATOMY OF HEART

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6. General.—The fully developed heart is a conical muscular pump inclosed in the pericardium, situated obliquely between the lungs, with its base upward and to the right and its apex downward and to the left. The average weight of the heart in adult males is 300 grams while in adult females it is somewhat less, 250 grams. In size it measures roughly 125 millimeters in length, 85 in width, and 60 in thickness, generally being considered as the size of the doubled fist.

7. Surface markings.—The heart is represented upon the chest surface by the following boundaries: The upper limit is demarcated by a line drawn from the lower border of the second left costal cartilage to the upper border of the third right costal cartilage. The lower limit is marked by a line drawn from the seventh right articulation to the apex in the fifth left interspace, 9 centimeters from the midsternal line. The right border extends from the third costal cartilage to the seventh right costosternal articulation and the left border from the second left costal cartilage to the apex. If the heart could be seen through the anterior chest wall, the right ventricle, the right auricle, the great vessels, and but a fringe of the left ventricle extending to the left of the right ventricle would be seen primarily.

8. General description.—*a.* If the heart is visualized as a somewhat flattened cone with one fairly sharp edge, the following anatomy is more readily understandable: The heart presents an apex, a base, two borders, and two surfaces. The apex is down, anterior, and to the left while the base is up, posterior, and to the right, extending beyond the midline. The apex is composed entirely of the tip of the left ventricle. The base is composed of the two auricles and the great vessels. The right border is long, thin, and much sharper than the left, which is short, rounded, and thick. The right border is made up of the right ventricle and a very small portion of the left ventricle near the apex whereas the left border is made up entirely of the left ventricle. The posterior surface is flattened and rests on the convexity of the diaphragm, whereas the anterior surface is rounded, convex, and considerably larger. The posterior or diaphragmatic surface is formed chiefly by the left ventricle while the anterior or sternocostal surface is formed chiefly by the right ventricle and a small portion of the left ventricle.

b. The heart is subdivided by a longitudinal musculomembranous septum into two halves, which are called right and left respectively. Each half is again subdivided transversely by a fenestrated muscular septum into two chambers, thus forming the four chambers of the heart. The two at the upper portion are termed right and left auricles while the two at the lower portion are termed right and

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left ventricles. The right side of the heart is venous while the left side is arterial.

c. The positions of the dividing septa are indicated on the heart surfaces by corresponding grooves. The sulcus between the auricles and ventricles is known as the auriculoventricular groove or coronary sulcus. The sulcus between the auricles, which is incomplete and present only near the ventricles, is known as the interauricular groove. Its continuation between the ventricles, which is complete and well marked, is known as the interventricular groove. The interventricular groove is divided into anterior and posterior portions, the sharp right border marking the division point.

9. Right auricle.—*a.* The right auricle is made up of two connecting chambers, the principal one being known as the sinus venosus and the minor one as the appendix auricula. The sinus or main cavity is situated between the superior and inferior vena cava and is connected below and anteriorly with the right ventricle. Its internal border is the interauricular septum, in the approximate center of which is a noval depression known as the fossa ovale. The fossa ovale is the closed remnant of the foramen ovale of the foetal heart. The appendix auricula is a small conical muscular pouch projecting from the sinus venosus forward and to the left side, overlapping the base of the aorta.

b. The capacity of the right auricle is approximately 163 cubic centimeters and its muscle walls average 2 millimeters in thickness.

c. The internal surface of the right auricle is smooth except in the appendix and the adjacent portion of the sinus venosus. Here it is thrown into parallel muscular ridges known as musculi pectinati. These ridges have the function of directing flow of blood in and out of the appendage.

d. The openings of the right auricle are superior and inferior venae cavae, coronary sinus, foramini Thebesii, and the auriculoventricular opening. The superior vena cava opens into the upper and posterior portion of the sinus venosus and has the function of returning the venous blood from the upper half of the body. The inferior vena cava opens into the lower and posterior portion of the sinus venosus near the auriculoventricular septum. Its function is to return the venous blood from the lower half of the body and is therefore slightly the larger of the two. The opening of the inferior vena cava is guarded on its anterior border by the incomplete and incompetent rudimentary valve of the vena cava, known as the eustachian valve. The opening of the superior vena cava has no such valve. The coronary sinus opens into the auricle between the in-

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ferior vena cava and the auriculoventricular opening. Its function is to return venous blood from the heart wall itself and its opening is protected by a semicircular fold of endothelium called the coronary valve. The foramini Thebesii are the mouths of small veins on various parts of the inner surface of the auricle. These small veins also return blood from the heart muscle. The auriculoventricular foramin connects the auricle with the right ventricle and is guarded by the tricuspid valve.

10. Right ventricle.—*a.* This cavity is trangular in form and extends from the auricle to near the apex of the heart. Its anterior surface is rounded and convex and forms the greater portion of the anterior surface of the heart. Its lower surface is flattened and rests upon the diaphragm and forms only a small portion of the posterior surface of the heart. Its posterior surface is formed by the interventricular septum, the surface of which bulges into the cavity of the right ventricle. This is due to the fact that the left ventricle is the thicker walled and being more powerful assumes the line of least resistance to become circular in cross section. The right ventricle being thinner walled and weaker is hung on the right side of the left ventricle and thereby assumes a semilunar shape in cross section. The upper, and thinner angle of the right ventricle forms a conical pouch, known as the infundibulum, from which the pulmonary artery arises.

b. The capacity of the right ventricle is given by White as 137 cubic centimeters and its walls average approximately 4 millimeters in thickness. The infundibular wall is somewhat thinner than the wall at the apical portion.

c. The openings of the right ventricle are the auriculoventricular and the pulmonary artery.

(1) The auriculoventricular opening is an oval orifice, situated at the base of the ventricle near the right border of the heart. Its circumference is given as 12 centimeters. It forms the means of communication between the right auricle and ventricle. The opening is surrounded by a dense fibrous ring covered with endothelium and is guarded by the tricuspid valve. This valve consists of three segments of triangular shape formed by reduplication of the endocardial lining of the heart and strengthened by a layer of fibrous tissue. These segments are connected at their bases to the auriculoventricular ring and meet in the center of the lumen when in the closed position. The ventricular surfaces afford attachments for the chordae tendineae which run from the free edge of the valve leaflet to the ventricular wall.

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(2) The pulmonary arterial opening is a more nearly circular orifice, approximately 9 centimeters in circumference, situated at the superior and anterior portion of the infundibulum. The opening is less sturdily reinforced by a fibrous tissue ring. The junction is guarded by the pulmonary valve, the three segments of which are semilunar in character. They consist of three semicircular folds, formed by reduplication of endocardium strengthened by a layer of fibrous tissue. They are attached by their convex edges to the wall of the artery at its junction with the ventricle, the straight edges being directed upward in the course of the artery, against the walls of which they are pressed during the passage of blood through the vessel.

d. The internal surface of the right ventricle, unlike that of the auricle, is roughened by the presence of a series of muscular projections resembling rugae. They fall into two types, the musculi papillari and the trabeculae carneae. The musculi papillari are ovoid muscle masses projecting into the ventricles, to the tips of which are attached the muscular insertion of the chordae tendineae. The trabeculae carneae are rugose masses of muscle tissue laid in either columns or loops just beneath the endocardium. Their function is said to be twofold, that of directing flow of blood into the infundibulum and that of strengthening the cavity wall. The most prominent of the loops is the moderator band which runs across the cavity, being connected at one end to the interventricular septum and at the other to the sternocostal wall. Its function obviously is to prevent over distention of the ventricle.

11. Left auricle.—*a.* This cavity, like the right auricle, consists of two parts: the sinus or principal cavity and the appendix auricula. The sinus is cuboidal in form and lies behind the aorta and pulmonary artery. Internally it is separated from the right auricle by the interauricular septum. The pulmonary veins empty into the posterior portion of it. The appendix is constricted at its junction with the rest of the auricle and is much more deeply indented than the right appendix. Its direction is forward and to the right and it overlaps the pulmonary artery.

b. The capacity of the left auricle is given by White as 140 cubic centimeters. Its walls are somewhat thicker than those of the right auricle, averaging 3 millimeters in thickness.

c. The following anatomical structures are to be noted in the left auricle: the openings of the pulmonary veins, the auriculoventricular opening, and the musculi pectinati. The pulmonary veins, four in number, empty two into the left side and two into the right side of the

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auricle. They are not provided with valves. The auriculoventricular opening is the means of communication between the left auricle and ventricle and is smaller than the corresponding one on the right side. As in the right ventricle the wall is smooth except in the appendage where the musculi pectinati run across its inner surface and the adjoining portion of the wall of the sinus.

12. Left ventricle.—*a.* This cavity is larger and more conical than the right ventricle and forms most of the posterior surface of the heart and all of the apex. Its walls are three times as thick as those of the right ventricle and bulge into the right ventricular cavity. The cavity of the left ventricle, called the aortic vestibule, presents a structure similar to but smaller than the infundibulum. This vestibule is not contractile but serves as a funnel directing the blood into the aorta.

b. The capacity of the left ventricle is approximately 121 cubic centimeters and its wall will average 11 to 12 millimeters in thickness. As with the infundibulum of the right ventricle, the aortic vestibule is constructed of somewhat thinner musculature than the main cavity.

c. The openings of the left ventricle are two in number, the auriculoventricular and the aortic.

(1) The auriculoventricular opening is similar in size (averaging 10 centimeters in circumference) and shape to that of the right ventricle, differing only in that the reinforcing fibrous ring is considerably stronger. It is guarded by the mitral valve. This valve is distinguished by the fact that it has only two leaflets, a larger anterior and a smaller posterior. The valve is a reduplication of the endocardium strengthened by fibrous tissue and is larger in size, thickness, and strength than the tricuspid valve. The mitral valve flaps are also furnished with chordae tendineae.

(2) The aortic opening with its yet heavier fibrous ring is situated to the front and right of the auriculoventricular orifice. The opening is somewhat smaller than the corresponding one on the right and measures from 7 to 8 centimeters in circumference. This orifice is guarded by three semilunar valves, one in front and two located posteriorly, which are reduplications of the endocardium strengthened by fibrous tissue, the aortic valve. These are the strongest and thickest valves of the heart.

d. The internal surface of the left ventricle presents the same type of structure as does the right ventricle. However, there is no prominent individual trabeculus such as the moderator band.

13. Valves.—*a.* The four major valves of the heart are divided into two groups according to their structural characteristics, the auric-

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uloventriculars and the semilunars. The two auriculoventricular valves are the tricuspid guarding the right auriculoventricular opening and the mitral guarding the left auriculoventricular opening. The tricuspid has three leaflets triangular in shape, namely, anterior, posterior, and medial according to their respective positions. The mitral valve has two leaflets of unequal size, the anterior being the larger, the posterior the smaller. All five leaflets have the common characteristics of being reduplications of the lining membrane of the heart reinforced by fibrous tissue, and of being reinforced by anchorages to the ventricular walls, the chordae tendineae. Chordae tendineae are numerous small fibrous cords attached at one end to the free margin of the valve and at the other end to the musculae papillari of the ventricular wall. Their mechanical function is to insure firm closure of the valve surfaces during systole by preventing the valves from being inverted into the auricle by the back pressue of the blood. In closing, these valves balloon out much like a sail which is filling with wind and which is held in a useful position by guy ropes (chordae tendineae). During diastole they flatten out against the ventricular wall as the blood flows past into the dilating ventricle.

b. The two semilunar valves are the pulmonary guarding the pulmonary orifice, and the aortic guarding the aortic orifice. Both have three cusps. The pulmonary cusps are designated right anterior, left anterior, and posterior. The aortic cusps are designated right, left, and posterior. During systole the cusps are folded against the vessel wall, allowing the outpouring blood to rush past unimpeded. During diastole (when the semilunar valves are closed) the individual cusps themselves are open, the opposing edges in tight contact, preventing back flow of blood into the ventricle. During closure the cusps form a pocket with the vessel wall and their action is like that of a pocket filling with fluid and distending. In distention the valves are in closing apposition. If the valve is visualized as a pocket, empty and folded against the vessel wall during systole, open and filled with blood by the back-pressure during diastole, its manner of functioning will be clear. The cusps are reduplications of endothelium reinforced by fibrous tissue and having additional thick reinforcing strands on their full margins. In the aortic valve, the pockets formed by the cusps are known as the sinuses of Valsalva, and are of importance because from the right and left sinuses the two coronary arteries arise.

c. The so-called minor valves of the heart are two in number, the coronary valve and the valve of the inferior vena cavae, both in the right auricle. They are merely folds of endocardium placed at the margins of their respective orifices, and act as incompetent unicuspid flap valves.

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14. Blood supply.—*a.* The blood supply to the heart itself is handled by two coronary arteries named right and left. The right coronary artery arises from the right sinus of Valsalva and passes forward between the pulmonary artery and the right auricle. It courses posteriorly in the right half of the auriculoventricular groove to the junction of the posterior interventricular groove, where it anastomoses with the circumflex branch of the left coronary artery. Two main branches are given off during its course, the right marginal and the posterior descending. The right marginal branch passes down the sharp right border two-thirds of the distance to the apex. The posterior descending branch leaves the main artery near its anastomosis and passes down the posterior interventricular groove to the apex. Several smaller branches and twigs are given off to the right auricle and to other adjacent structures along the course of the artery and its branches. The left coronary artery arises in the left sinus of Valsalva and passes forward between the pulmonary artery and the left auricle. At the auriculoventricular groove it divides into two main branches, the circumflex and the anterior descending. The circumflex passes posteriorly in the left half of the auriculoventricular groove to anastomose with the right coronary artery. The anterior descending passes down the anterior interventricular groove to the apex giving off smaller branches of twigs along its course. The circumflex gives branches to the left auricle. The left margin has no artery distinctively its own.

b. The right coronary artery supplies the right auricle, approximately the posterior two-fifths of both ventricles, and a corresponding portion of the interventricular septum. The left coronary artery supplies the left auricle, the anterior three-fifths of both ventricles, and a corresponding portion of the interventricular septum. The terminal network of both vessels anastomose freely, the importance of which fact is brought out later. It is only fair to point out, however, that one very important school of anatomists holds that there are no coronary end anastomoses in a normal heart. This group holds that such anastomoses develop only in coronory sclerosis or occlusion.

c. The venous return from the cardiac structures is carried through the coronary veins. The veins roughly approximate the arteries both in course and designation, and empty into the right auricle via the coronary sinus.

15. Nerve supply.—*a.* The heart is governed by two opposing sets of extrinsic nerves, the vagi and the sympathetics. The vagi arise in the medulla and furnish both sensory and motor fibers. The

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sympathetics arise from the rami communicantes of the cervical and upper thoracic nerves. Both vagi and sympathetics reach the heart through the superficial and deep cardiac plexuses. The superficial cardiac plexus lies beneath the arch of the aorta and is formed by the superior cardiac branch of the left sympathetic and the lower cervical cardiac branch of the left vagus. The deep cardiac plexus lies in front of the bifurcation of the trachea and is formed by the cardiac fibers of the cervical sympathetic ganglia and the cardiac branches of the vagus. The two branches mentioned as forming the superficial plexus are the only two not reaching the deep plexus.

b. In general, the right vagus and sympathetic supplies the right half of the heart and the left vagus and sympathetic supplies the left half of the heart. However, there is free intercommunication between the two. The right vagus and sympathetics supply specifically the sino-auricular node and the left vagus and sympathetics supply the auriculoventricular node. Sensory fibers are distributed over the heart by two minor plexuses called the coronary plexuses, right and left following the course of the right and left coronary arteries respectively.

c. The function of the vagus supply to the heart is inhibitory, whereas that of the sympathetics is one of acceleration. Stimulation of the vagus slows the heart rate and diminishes the force of contraction. Stimulation of the sympathetics increases the rate and force of the heart beat. Between them they control the action of the heart and transmit information to the heart as to the body's circulatory requirements. It is only through these opposing sets of nerves that a circulation adequate to meet the changing demands of the body can be met.

16. Conduction system.—The intrinsic nerve supply to the heart is known as the conduction system and is concerned with origination and transmission of activating impulses throughout the heart. It is composed of five parts, the sino-auricular node, the auricular muscle, the auricular ventricular node, the bundle of His, and the Purkinje system. The sino-auricular node, also known as the pacemaker or Keith-Flack node, is located at the junction of the superior vena cava and right auricle laterally in the sulcus terminalis beneath the epicardium. It is a 10-millimeter bundle of specialized neuromuscular tissue from which the normal rhythmic impulses of the heart arise. The auricular muscle, although not neuromuscular in character, conducts the impulse across its substance to the auriculoventricular node. This node, also known as the node of Tawara, is located in the right auricle at the posterior and lower part of the interauricular

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septum. This node is similar in size and structure to the sino-auricular node, varying somewhat in function in that it transmits the normal impulse rather than initiates it. The bundle of His is a narrow flat band of neuromuscular tissue, continuous with the auriculoventricular node, running 1 centimeter forward and down along the right side of the interauricular septum to the musculomembranous junction. Here it divides into two branches, the right and left bundle branches. The right branch passes beneath the tricuspid valve, down along the interventricular septum, reaching nearly to the apex of the ventricle before it begins its fanlike arborization. The major subdivision of the right branch crosses the moderator band to reach the anterior wall of the right ventricle from which point it begins its arborization. The left bundle branch passes through the membranous septum and almost immediately begins the subdividing process, having a very short course as a single undivided nerve. The Purkinje system is a collection of neuromuscular cells which act as the end organs of the conduction system. They are located subendocardially and are so distributed that every myocardial cell unit in the ventricles is supplied by Purkinje cell fibrils.

17. Special anatomy.—*a.* The functional anchorage of the heart is located in the auriculoventricular septum. Here is situated the framework against which the cardiac musculature pulls during contraction. This framework consists of the trigonum fibrosum and the fibrous rings surrounding the valve foramina. The trigonum fibrosum is a heavy triangular mass of fibrous tissue located between the two auriculoventricular openings and the aortic ring. Its prime function is that of a reinforcing structure which maintains the valve rings and chambers in their proper relative positions, and to which the three valve rings mentioned are anchored. The musculature of the auricles and ventricles receives its origin and insertion in the trigonum fibrosum and its adjacent valve rings. The systemic aortic and the pulmonary artery arise from and are anchored to the aortic and pulmonary rings respectively.

b. The muscle fibers of the heart are arranged in sheets or strands according to the lines of force executed in contraction. In the auricles these fibers fall into two groups, deep and superficial. The deep fibers arise at the auriculoventricular rings, encircle their individual auricle, and reinsert in the same valve ring. The superficial fibers arise at the ariculoventricular rings, encircle both auricles, and reinsert at the same ring. Both groups send off fibers which encircle the great veins of both auricles and which, during contractions, exert a sphincter action to somewhat restrict the backward flow of blood from the auricles. In

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the ventricles the muscle bundles become much more complex but again fall into deep and superficial layers. The deep fibers arise at the auriculoventricular rings, encircle their individual ventricles, and end in the papillary muscles, columnae carnae, or reinsert into the structures at the ventricular base. The superficial fibers fall into two distinct bands, the sinospiral and the bulbospiral. The sinospiral arises in the tricuspid region and spirals about the right ventricle, being especially prominent near its apex. The bulbospiral arises at the aortic and mitral rings and encircles the left ventricle, being especially prominent near its apex. The spirals themselves are made up of a deep and superficial layer, running at right angles to each other. Muscle contraction in the heart proceeds along these muscle bundles, each pulling against the reinforcing structures in the auriculoventricular septum, forcing the blood along its course by their squeezing action. Thus the heart muscle, unattached to bony structure as is most voluntary muscle, is enabled to pull against solid structures within itself in accomplishing its physiological purpose of propelling the blood, a sort of lifting itself by its bootstraps.

18. Pericardium.—The pericardium is the outer protective covering of the heart and the roots of the great vessels. Its function is to permit the contractual movements of the heart to occur without friction or contact with the other structures of the mediastinum and thorax. It consists of two portions, the fibrous sac and the serous sac. The fibrous sac is a thick, heavy membrane arising from the outer coats of the great vessels and completely enclosing the heart. It is maintained in position by three attachments; to the great vessels above, to the sternum by the sternopericardiac ligaments, and to the diaphragm by a central tendon. The serous sac is a reduplicated serous membrane, the parietal portion of which is adherent to and lines the fibrous sac, and the visceral portion of which covers the myocardium. Frictionless movement of the heart is maintained by the contact of these two serous surfaces.

19. Aorta.—The aorta is the large vessel conducting arterial blood from the heart to the systemic circulation. It is divided for purposes of description into three portions, the ascending aorta, the transverse aorta, and the descending aorta. The ascending aorta is of most importance to cardiac pathology. It is roughly 5 centimeters in length and arises at the upper portion of the left ventricle on a level with the lower border of the third costal cartilage. It passes obliquely forward and upward and to the right. The only branches given off are the two coronary arteries. The transverse aorta, also known as the arch, is roughly 5 centimeters in length. Its course is

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that of an arc, directed generally backward toward the left side of the trachea. The lower limit is described as being the upper border of the right third costosternal articulation and its upper limit is described as being 5 centimeters below the upper border of the sternum. The branches arising from this section of the aorta are the innominate, the left common carotid and the left subclavian. The descending aorta begins at the lower border of the fourth dorsal vertebra and passes down to the lower border of the fourth lumbar vertebra where it divides into the common iliac arteries. The descending aorta is divided into two portions at its diaphragmatic hiatus, a thoracic portion and an abdominal portion. From these two portions arise all of the important visceral branches. The aorta is 3 centimeters in diameter at its origin and roughly 1.75 centimeters in diameter at its abdominal bifurcation.

20. Recommended texts.

Textbook of Anatomy (Cunningham)	William Wood and Co.
Gray's Anatomy (Lewis)	Lea and Febiger.
Heart Disease (White)	Macmillan.
The Mechanism and Graphic Registration of the Heart Beat (Lewis)	Shaw and Sons, Ltd.
The Blood Supply of the Heart (Gross)	Hoeber.
The Heart as a Power-Chamber (Sainsbury)	Oxford Publications.
The Heart (Neuhof)	Blakiston.

Section III

HISTOLOGY OF HEART

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21. Endocardium.—The entire inner surface of the heart is covered by the endocardium, which forms the serous lining of all the chambers and is continuous with the intima of the blood vessels. It consists of two layers, the inner composed of a single layer of serous cells and the outer, connecting the serous layer to the myocardium, composed of fibrous and elastic tissue. The endocardium is entirely devoid of blood vessels.

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22. Myocardium.—The myocardium is made up of a type of tissue unlike any other of the body, interlacing striated involuntary muscle. Each fiber is a short cylinder, one-third smaller than voluntary fibers, with a single centrally placed nucleus. The striations are both longitudinal and transverse, and are somewhat less easily seen than similar structures in voluntary muscle. The longitudinal striae are fibrillae, and transverse, intercalated disks. Each fiber subdivides and branches at both ends, each branch anastomosing with a similar subdivision of some neighboring fiber. The nature of this anastomosis is not entirely clear and it is a question whether each cell is a distinct unit or the entire heart muscle a syncytium. As a result of this branching anastomosis, heart muscle cells do not lie parallel as do the cells of other muscle types but are complexly interlaced and intertwined. The secret of the heart's greater efficiency is believed to lie in its peculiar muscle structure.

23. Pericardium.—The pericardium is best described as two sacs, one inside the other, enclosing the heart. The outer is the fibrous sac, and the inner the serous sac. The fibrous sac which functions as a protective membrane is a dense sheet of fibrous tissue interlaced with elastic cells. The serous sac is a thin serous membrane. It is reflected back on itself at the roots of the great vessels to form two opposing layers, the visceral or epicardium, and the parietal. The epicardium is closely bound to the myocardium by loose fibrous tissue, whereas the parietal layer is closely bound to the fibrous sac. The function of the opposing serous surfaces is to allow frictionless movement of the heart within its fibrous protection.

24. Valve structure.—All cardiac valves are reduplications of endocardium strengthened by an intermediate supporting stratum of fibrous tissue. The leaflets of the four major valves are heavily reinforced with fibrous tissue on their free edges and at the line of closing apposition. The semilunars are so heavily reinforced as to warrant a special nomenclature. In the center of the free margin of each semilunar cusp is a heavy nodule of fibrous tissue called the nodulus valvulae semilunis. Extending in both directions from this nodule along the free edge of the valve cusp to the vessel wall is a heavy band of fibrous tissue called the lunule. Other smaller bands of fibrous tissue radiate fanlike from the nodule to the attached margin at the valve rings. The auriculoventricular valves are not so well reinforced intrinsically but receive an equivalent amount of support extrinsically by means of the chordae tendineae. All major valve leaflets are attached to the dense fibrous rings which reinforce the four major valve foramina. The size and density of this reinforcing

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tissue vary with the pressure load each valve carries, being much heavier on the left side of the heart.

25. Conduction system.—*a.* The conduction system is histologically distinct from the myocardium, although structurally interwoven with it. Both the sino-auricular and auriculoventricular nodes have essentially the same structure. Both are composed of an interlacing network of slender, longitudinally striated neuromuscular fibers, one-third the size of the heart muscle fibers. Their nucleii are elongated and centrally placed. These cells are interlaced in a plexiform manner and embedded in a closely packed connective tissue mass.

b. The fibers in the bundle of His are similar to those of the nodes except that they are arranged in a parallel manner and the striations are much less distinct.

c. The Purkinje system is composed of a yet different type of neuromuscular cell; one with granular protoplasm, irregular transverse striation, and peripherally placed nucleii. They are somewhat larger than myocardial cells and more or less ovoid in appearance. These cells connect with one another at their extremities but do not branch. They lie just under the endocardium and are intimately bound to the myocardial fibers. They are the end organs of the conduction system, transmitting the activating impulse to each individual muscle cell.

26. Recommended texts.

Textbook of Anatomy (Cunningham)..... William Wood and Co.
Gray's Anatomy (Lewis)..... Lea and Febiger.
The Mechanism and Graphic Registration of the Heart Beat (Thomas Lewis)..... Shaw and Sons, Ltd.
Textbook of Histology (Bremer)..... Blakiston.

SECTION IV

PHYSIOLOGY OF HEART

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27. Cardiac function.—*a.* The ultimate function of the cardiovascular system is to maintain an effective circulation of blood throughout the entire body. This is accomplished by propelling blood in the needed quantities through the two circulatory systems of the body, the systemic and pulmonary circulations. The systemic circulation carries oxygenated blood to the tissue capillaries and drains venous blood back into the heart. The pulmonary circulation carries venous blood to the pulmonary capillaries for oxygenation and drains the newly oxygenated blood back into the heart for redistribution throughout the systemic circulation.

b. In order to maintain an adequate supply of blood flowing through the two circulatory systems, the heart undergoes a regular series of contractions at an average rate of 72 per minute. Each beat is a complicated series of expulsive muscular activity plus retentive valvular action. The events comprising each beat are known as the cardiac cycle.

28. Cardiac cycle.—*a.* A cardiac cycle is designated as the series of events comprising the heart beat, beginning at any given feature of that series and ending just before the next appearance of that feature. In general the cycle consists of the simultaneous contraction of the auricles, followed immediately by the simultaneous contraction of the ventricles, and completed by a period of muscular rest. The contraction of either auricle or ventricle is known physiologically as its systole and the rest period is known as its diastole. During diastole the musculature becomes oxygenated, nourished, and is relieved of the CO_2 and metabolites produced during the contraction. The duration of auricular systole is classically given as 0.1 second; of ventricular systole as 0.3 second; and of diastole as 0.4 second. Clinically, systole is usually understood to be the ventricular contraction and the rest of the cycle is known as diastole. The auricular contraction is referred to clinically as the presystolic phase of diastole.

b. A detailed description of the events comprising a cardiac cycle is best begun in diastole. During diastole both the auricles and the ventricles are passively filling with blood flowing in from the great veins. The auriculoventricular valves are open and there is free communication between the auricles and ventricles. Backflow from the systemic and pulmonary circulations is prevented by the closed semilunar valves. Auricular systole forces the auricular contents into the ventricle, distending the latter. Backflow into the great veins is retarded somewhat, but not entirely prevented, by the contraction of the muscular rings about their mouths. Following the completion of auricular systole the ventricles begin their contraction.

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As the interventricular pressure rises, the auriculoventricular valves are forced together, closing off backflow into the auricles. As the pressure continues to rise and finally exceeds that in the great arteries, the semilunar valves open and the ventricular contents are ejected into both the pulmonary and systemic circulations. As ventricular contraction ends and the interventricular pressure falls, the excess of pressure in the great arteries closes the semilunar valves, preventing backflow into the ventricles.

c. During the time that the ventricles have been contracting, the auricles have been in their diastolic phase, the relaxed cavities receiving free flow of blood from the veins. This flow is aided by the negative pressure created by the relaxation of the auricular muscle. As the auricles fill and the interauricular pressure exceeds that of the relaxing ventricles, the auriculoventricular valves open and blood again flows freely between the two chambers. The active function of the auricles is to collect blood during the ventricular systole and to force that blood into the ventricles under pressure, thus dilating the chamber and stretching the musculature for more efficient contraction. The function of the ventricles with their much heavier muscle wall is to drive blood to the tissues and lungs which is accomplished by overcoming the peripheral resistance. The function of the valves is to direct the flow of blood, which is accomplished by preventing backflow in the undesired direction.

29. Output of heart.—*a.* The output of the heart per minute is the main factor which determines the circulation rate. This output varies from 3 liters a minute during rest to 20 liters or more during exercise. It depends on two factors, the output per beat and the rate per minute. The output per beat depends upon the diastolic filling, the competency of the valves, and the contractile power of the heart muscle. Diastolic filling is of importance because the heart cannot discharge during systole more blood than it receives during diastole. The competency of the valves determines whether or not all of the blood will be propelled forward or whether some will also be forced backward (as in mitral insufficiency). The status of the heart muscle determines whether or not the propelling force is adequate.

b. Diastolic filling depends further upon the venous pressure and venous inflow. Adequate venous pressure insures adequate flow of blood along the venous tree. Venous inflow is regulated by the action of the skeletal muscles which mechanically propel the blood through the muscles and into the larger veins. In addition, the movements of the diaphragm, by raising the introabdominal pressure, aid in keep-

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ing up an adequate venous pressure, and hence an adequate filling of the heart.

c. Aside from pathological factors, the contractile power of the heart muscle depends on three physiological factors: the amount of stretching prior to contraction, the size, and the nutrition of the muscle. The amount of stretching depends upon the volume of diastolic filling, and the effect of this stretching is known as "the law of the heart," which is "the force with which a muscle contracts is directly proportional to the length of the fibers just before they begin to contract." The contractile power of the heart also depends both upon the size of the musculature and upon its nutritive condition. All other conditions being constant, a well-nourished, thick-walled ventricle will contract more strongly than a thin-walled, ill-nourished one. This can explain the difference in the so-called reserve power of the heart in different individuals. It also explains the difference in exercise tolerance between athletes and those doing sedentary work.

d. Thus it is seen that the output of the heart per minute is the product of its output per beat and of the pulse rate. The output per beat (venous pressure being adequate) depends largely on the contractile power of the heart. It is obvious, then, that the greater the output per beat, the less acceleration of the pulse required to maintain an adequate output per minute; on the other hand, the smaller the output per beat, the greater the acceleration of the pulse necessary to maintain that output. In this way acceleration of the pulse can to a certain extent compensate for lessened contractile power of the heart muscle. However, the efficiency of the heart diminishes if the acceleration of the pulse is unable to make up for the lessened contractile power. Heart failure results when the heart is unable to maintain a normal output per minute under conditions of rest, or is unable to increase its output in response to demands made upon it.

30. Fundamental properties of heart muscle.—*a.* Heart muscle has at least four functions: Stimulus production, stimulus conduction, contractility, and irritability. In other words, the heart has the property of initiating spontaneous beats (stimulus production); of conveying stimuli to various portions of the heart (stimulus conduction); the power of receiving stimuli and becoming excited thereby (irritability); and finally, the power of responding by contraction (contractility). The exact mechanism that initiates such beats is not understood, but it is generally assumed that after each cardiac contraction there is a gradual accumulation of certain unstable compounds in the heart muscle, which finally reaches a point where these unstable compounds break down suddenly and, in doing so, initiate a contraction.

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b. The above four properties have a refractory period (period of inexcitability), that is, after they have exercised their function they must remain quiescent until certain reconstructive changes have taken place before they are capable of responding to a stimulus again. During this period they are said to be refractory to a stimulus. This refractory period begins when the excitation wave enters the heart muscle and lasts to about the end of systole. After this, the excitability returns rapidly. The refractory period plays an important role in the analysis of certain of the cardiac arrhythmias, because a stimulus which reaches a chamber during its refractory period produces no systole.

c. The term "tone" used in connection with heart muscle has been considered by clinicians to mean a state of slight continued contraction even during diastole. Starling, however, is against the existence of diastolic tone. He believes that the heart muscle is normally completely relaxed during diastole, and uses the term "tone" to indicate the physiological fitness of the muscle fibers. That is, a well-nourished muscle will contract more strongly than a poorly nourished one. Tone, therefore, means greater force of contraction during systole, not lessened relaxation during diastole.

d. Although any portion of the heart muscle is inherently capable of initiating contractions, the pace of the whole heart is usually governed by impulses arising in the sino-auricular node. In case, however, the stimulus from the sino-auricular node fails to reach the conducting system of Tawara, the auriculoventricular node, or other portion of the bundle of His, is capable of initiating stimuli, but usually at a much slower rate than the normal one. The function of the pacemaker will also pass to the auriculoventricular node whenever the automatic rate of the latter exceeds the automatic rate of the sino-auricular node.

e. The spread of this impulse from the pacemaker to all portions of the heart muscle can be recorded by the electrocardiograph. This records the variation in the electrical potential of the heart. Clinically, it is important to know whether the cardiac functions of stimulus production, stimulus conduction, and contraction proceed in a normal fashion. Whenever this does not occur, cardiac irregularities with consequent impairment of circulatory efficiency occur.

31. Conducting system.—*a.* The mammalian heart is normally excited by impulses formed at the sino-auricular node. The exact nature or causation of these impulses is unknown, although it is presumed to be electrochemical in action. The impulse spreads through the walls of the auricle in an ever-widening circle, stimulating the

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auricular musculature to contraction. When it reaches the outer border of the auricular tissues, the impulse is picked up by the auriculoventricular node of Tawara. This node transmits the impulse to the bundle of His, through which it passes into both bundle branches and into the Purkinje system. Through the Purkinje system the impulse is transmitted to each and every cell in the ventricular musculature, stimulating them to contraction.

b. The speed of travel of the impulse through the various structures is given as follows:

	Millimeters per second
Auricular musculature	1,000
Auriculoventricular node	400
Bundle of His and Purkinje system	4,000

32. Extrinsic nerve supply.—*a*. The rate of production of the impulse of the sino-auricular node is influenced by the two opposing extrinsic nerve supplies. The sympathetics furnish accelerator control and the vagus supply furnishes inhibitory control. The needs of the body are communicated to the heart through these two systems, and the rate at any given moment depends upon which nerve supply system is dominant at that particular time. Impulses through these nerves modify the fundamental properties of heart muscle, but do not cause contractions of the heart directly. In a general way, stimulation of the vagus depresses impulse formation and conduction, whereas stimulation of the sympathetic acts in an opposite manner. The normal rate of impulse formation is given as 72 beats per minute.

b. The carotid sinus, mentioned frequently in the newer literature, is a very definite portion of the depressor mechanism. This sinus is located in the bifurcation of the common carotid and has as its primary function control of the carotid circulation. However, stimulation of this sinus by digital pressure causes a fall in blood pressure and a reflex cardiac inhibition, often entirely stopping the heart for several seconds. Its clinical application is not yet fully understood.

33. Heart sounds.—Each cardiac cycle is accompanied by two distinct sounds and in some cases by a third sound. The first, occurring at the beginning of ventricular systole, is made up of two elements, namely, the vibrations caused by the closure of the auricular valves and the sound produced by the contracting ventricles. The second sound, occurring at the beginning of diastole, is produced by the closing of the aortic and pulmonary semilunar valves. The third heart sound is sometimes heard at the apex of the heart in normal individuals. This sound is faint and dull and follows immediately after the second sound. It is probably due to a movement of the

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auriculoventricular valves as the blood is rushing into the ventricles in early diastole. This is found in many normal young persons.

34. Coronary circulation.—The coronary circulation is usually considered a part of the systemic circulation. The flow of blood through the coronary arteries is distinct in that only during diastole do the arteries fill up and deliver their contents to the muscle capillaries. During systole the semilunar cusps of the aortic valve are forced against the aortic wall and the sinuses of Valsalva are empty of blood except during the very early phases of the valve opening. Hence the coronary arteries, arising as they do from the sinuses of Valsalva, receive no blood during systole. Further, during systole the muscle fibers shorten and obliterate the capillary interspaces, thus squeezing the blood into the coronary veins and promoting venous return into the right ventricle. Therefore, during systole the arteries receive no blood, the capillaries are empty, and venous flow is aided. During diastole, the aortic valve cusps are held in apposition by distention of the sinuses of Valsalva with blood from the aortic backflow. This backflow, created by the diastolic arterial pressure, forces blood into the arteries and into the capillary interspaces now opened by the relaxation of the musculature. Thus the coronary circulation is dependent upon the maintenance of diastolic pressure, a fact which is of considerable importance in the consideration of aortic insufficiency. Inasmuch as the heart muscle is nourished only during diastole, the efficiency of the heart depends also on the length of diastole. The longer diastole, the more thorough will be the perfusion of the capillary bed, and the shorter diastole, the less will be the amount of nourishment and oxygen to reach the muscle. Herein lies the difficulty engendered by the rapid rates, a shortening of the length of diastole.

35. Vascular apparatus.—*a.* The vascular apparatus which consists of a closed system of vessels, contains blood and helps transmit it to and from all regions of the body. It is divided into a systemic system which consists of a collecting and distributing system as mentioned, and a pulmonic portion. The arteries serve to transmit the blood ejected from the heart to the capillaries and by virtue of their elasticity they are capable of being distended and elongated and of recoiling and returning to their former condition. This enables them to adapt themselves to variations in the volume of blood discharged from the ventricles in a single beat. The contractility of the arteries permits of a variation in the amount of blood passing through a given capillary area in a unit of time. This tonic contraction of the arterioles increases the resistance to the outflow of blood into the

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capillaries, and thus assists in maintaining the blood pressure in the arteries. This resistance is generally termed the peripheral resistance.

b. The capillaries are interposed between the arterioles and venules, and it is through these capillaries that the necessary exchange of materials between the blood and tissues occurs. The ultimate function of the circulation is to provide an adequate distribution of blood under a proper pressure for this purpose. In order that nutritive materials may pass across the capillary walls in amounts sufficient to supply the tissues, it is essential that the blood shall flow into and out of the capillary bed constantly in volumes varying with the activities of the tissues under a given pressure and with a definite velocity. This condition is made possible by the action of the heart and vascular system.

36. Blood pressure.—*a.* The tension under which the blood is maintained within the vascular system is known as the blood pressure. It is that pressure which is exerted against the sides of the vessels by the blood stream. That within the arteries is the arterial blood pressure, while that within the veins is the venous pressure. The arterial pressure is that which is most commonly determined clinically.

b. The factors which determine the blood pressure are six in number: the force of the heart beat; the resistance to the flow of blood through the vessels, and especially the peripheral resistance in the region of the arterioles and capillaries; the elasticity and contractility of the arteries; the surface area of the blood vessel walls of the resistance to blood flow produced thereby; the blood viscosity; and the total blood volume.

c. The blood pressure will be raised (other factors remaining constant) if the heart forces more blood into the vascular system than escapes at the periphery of the arterial circulation, that is, if the output of the heart per minute is increased. This, in turn, depends on the output per beat, which is determined by the diastolic filling and the rate of heartbeat. The increase or decrease in peripheral resistance will markedly influence the blood pressure. The blood pressure is largely regulated by the contraction and dilatation of the arterioles. Under normal conditions they are kept in a state of tonic contraction, which may be increased or diminished according to the body needs, through impulses from the central nervous system.

d. A constriction (vasoconstriction) of these arterioles causes an increase in arterial pressure and a decrease in venous pressure. A dilatation (vasodilatation) has the opposite effect. Not only do the arterioles play an important role in regulating the systemic blood pressure, but the degree of their contraction governs the local blood flow

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to particular vascular areas. An increased blood flow to one part of the body may be associated with a diminished flow to another part. Dilatation of the splanchnic vessels is usually accompanied by constriction of the surface vessels and vice versa.

e. The elasticity of the arteries serves to convert the intermittent flow of blood from the heart into a constant current. The arteries dilate during cardiac systole to accommodate themselves to the increased blood volume, while during diastole they contract, thereby maintaining a certain blood pressure. A diminution in elasticity tends to interfere with the constancy of blood flow and the vessels become more or less rigid. When this occurs, as in arteriosclerosis, the systolic pressure is much increased and the diastolic pressure becomes lower.

f. The surface area of the blood vessel wall is to a considerable extent responsible for the height of the systolic pressure. While the resistance offered to the blood stream in the aorta is not large, it becomes so in the terminal divisions of the arterial system where the capillary bed is large.

g. Blood volume and blood viscosity influence blood pressure to some extent. A loss of blood will cause a fall in blood pressure as will a diminished blood viscosity. It is reasonable to think that the opposite condition causes a rise of blood pressure.

h. In the arteries the maximal pressure, which is the result of cardiac systole, is known as the systolic pressure; that occurring at the end of diastole, the diastolic pressure; and the difference between the two, the pulse pressure.

i. Blood pressure taken in modern clinical practice is understood to be that of the brachial artery or an artery of similar caliber. Blood pressure varies with the size of the artery, being much greater in the aorta than in the brachial, and much greater in the brachials than in the digitals. Further, the systolic pressure is from 20 to 40 millimeters mercury greater in the femoral arteries than in the brachials.

37. Recommended texts.

Textbook of Physiology (Howell)	Saunders.
Human Physiology (Starling)	Lea and Febiger.
Applied Physiology (Wright)	Oxford Publications.
The Heart as a Power Chamber (Sainsbury)	Oxford Publications.
Vital Cardiology (Williamson)	William Wood and Co.
Diseases of the Heart (Cowan and Ritchie)	Arnold.
Diseases of the Heart and Aorta (Hirschfelder)	Lippincott.

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CHAPTER 2

PHYSICAL EXAMINATION AND RELATED SUBJECTS

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SECTION I

CARDIOVASCULAR EXAMINATION

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38. General.—*a.* An adequate diagnosis of cardiac disturbances is based on a thorough examination of the entire cardiovascular system. The examination should be carried out in an orderly manner so that nothing of importance will be overlooked. The complete examination should take the greater part of an hour except in cases of obvious distress or decompensation. Here the more thorough procedure can be postponed until at least some degree of recovery has taken place.

b. A complete cardiovascular examination consists of four major procedures and numerous minor adjuncts. The four major procedures are—

(1) History.
(2) Physical examination.
(3) Electrocardiogram.
(4) X-ray of chest.

To these should be added, as and when indicated, such miscellaneous laboratory procedures as serology, urine examinations, blood counts, blood chemistry, sedimentation rate estimation, and basal metabolic rate. These minor adjuncts are of much importance and should not be overlooked as aids to diagnosis, although their routine use in each and every case is somewhat open to question. For detailed consideration of these procedures, see any good text on laboratory methods.

c. Likewise, electrocardiography, being a specialty in itself and having an extensive and complicated subject matter, will not be con-

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sidered in this manual. Both electrocardiography and the roentgenologic interpretation of cardiac conditions will be considered in a supplementary compilation.

39. History.—The history should include four sections: family history, past history, symptomatology, and past exercise tolerance.

a. Family history.—Family history is of relative importance only. The advantage of eliciting past cardiovascular diseases in ancestors and relatives lies entirely in the fact that heredity seems to be a factor in some cardiac conditions, especially those of the degenerative type. A tendency to hypertension, arteriosclerosis, coronary disease, and early myocardial degenerative changes seems to be inherited. Conversely, a sound and resistant cardiovascular system seems to be a heritage passed from one generation to another.

b. Past history.—(1) The past history is of importance because from this source of information, the etiology of the present illness is frequently determined. All past illnesses should be inquired into, the severity, number of attacks, and the resultant disability. This is especially true of the infectious diseases, scarlet fever, typhoid fever, diphtheria, pneumonia, and the streptococcal infections. These diseases all take their toll of the heart reserve, no matter how minor, due to the accompanying acute inflammations of the myocardium. Rheumatic fever is the most important single disease entity, and it should be inquired into in great detail. All of its various manifestations have a direct causative bearing on cardiac disease. Habits as to sleep, food, tobacco, and alcohol should be noted. In women, the obstetric and gynecologic history should be searched for possible etiological factors, especially puerperal sepsis. Venereal history is of importance in the middle-aged patient, especially in the colored races.

(2) History taking is an art, the technique of which is acquired only through considerable experience. It is good practice to have a form to follow, in order that nothing of importance be missed. It is equally bad practice to follow this form too rigidly in questioning. The procedure should be flexible enough to be varied with the indications and leads the patient furnishes. Especially in cardiovascular history is it well to allow the patient to talk about his troubles in whatsoever channel he selects, adding an occasional question here and there to keep the conversation within the limits which have a bearing on the disease. Much more information will be gained in this manner.

c. Symptomatology.—(1) Following the review of the past diseases, the symptoms of the present complaint should be inquired into.

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Cardiac pain, dyspnoea, cyanosis, and edema should head the list and each should be considered as to onset, duration, type, and severity.

(2) Cardiac pain is often of the greatest importance and its nature, radiation, severity, and relationship to such other factors as meals, exercise, and sleep should be ascertained. The most common cause of cardiac pain is disease of the coronary arteries. Other causes are acute pericarditis, aortitis, aneurysm, and rheumatic carditis. The pain of coronary disease is of the substernal variety and ranges from mild aches of short duration to the severe, excruciating pain of angina pectoris or coronary occlusion. Radiation in this type of pain is to the left shoulder, arm, wrist, and fingers. Less often it is referred to the right shoulder and arm, epigastrium, or abdomen. Infrequently, coronary type of pain can occur without its primary substernal location, and in this event, becomes very confusing. Aortitis and aneurysm produce a boring, aching, severe type of pain, which is usually centered in the upper chest and is referred to the back. Rheumatic carditis and acute pericarditis occasionally have accompanying pain of varying degrees of severity. This type of pain is precordial rather than substernal and like anginal distress is sometimes referred to the left shoulder.

(3) Dyspnoea accompanies congestive failure of all degrees and also complicates several of the pulmonary disturbances. Inquiry will differentiate this point if the possibility of pulmonary disease as a causative agent is borne in mind during questioning.

(4) While cyanosis and edema are not symptoms in the strictest interpretation of the word, they should be included at this point because both are usually an integral part of the pattern of serious myocardial pathology. Likewise, although hypertension is not a symptom, knowledge of it presence should be noted along with the mode of and reason for its discovery.

(5) After disposing of the four major features, the following symptoms, usually secondary effects of myocardial disease, should be noted:

(*a*) *Physical symptoms.*

Palpitation.	Constipation.
Nausea and vomiting.	Cough.
Gastritis and indigestion.	Production of sputum.
Liver disturbances.	Visual disturbances.

(*b*) *Nervous symptoms.*

Tinnitus.	Faintness.	Tremor.	Sweating.
Headache.	Giddiness.	Flushing.	Cerebral accidents.

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Any of these more general symptoms have a definite bearing on the determination of etiology, severity, and course of the disease. They are of importance in that their presence adds clarifying detail to the general pattern created by the four major symptoms. However, in a work of this scope, detailed consideration is omitted, and the student is referred to the more extensive standard texts for further information.

d. Past exercise tolerance.—Questions as to exercise tolerance complete the history. In order to evaluate properly this factor, the nature of the patient's occupation and normal exercise tolerance prior to the onset of the cardiac disorder must be learned. In many cases, a statement as to the patient's ability to climb a moderate flight of stairs, to walk on the level, or to do slight exercise should be obtained. In children, the parents can give a history as to the child's reaction to the ordinary games of childhood.

40. Physical examination.—After a thorough history has been obtained, proceed with the physical examination and, in order that no important factor be overlooked, it is advisable to follow a set routine, the one most commonly used being that of—inspection, palpation, percussion, and auscultation. To these classical procedures should be added a fifth; estimation of exercise tolerance as it exists at the time of examination. Blood pressure must be taken. The estimation of present exercise tolerance is a very important procedure which under no circumstances should be omitted in a thorough cardiac examination.

a. Inspection.—Inspection often reveals important indications as to the nature of the disease entity present. The following points should be especially looked for: apex beat, cyanosis, respiration, edema, pulsations of neck veins, exophthalmos, bulging of the precordium, abdominal pulsations, capillary pulsations, and clubbing of the fingers. The apical impulse is of especial importance in that cardiac enlargement, or the lack of it, is immediately indicated. Also, some slight indication of the contractile power of the heart may be obtained by ascertaining the character of the thrust against the chest wall. Cyanosis is checked for degree, color, location, and extent. The character of the respiration, whether shallow and rapid, deep and stertorous, normal or labored, indicates the extent and type of pulmonary involvement. The extent and type of edema should be noted as an index of the degree of congestive failure. Abnormal pulsations or engorgement of the neck veins above the level of the heart give one of the most important indices of increased venous pressure. Exophthalmos and other signs of Graves' disease may give

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an immediate clue to etiology. Bulging of the precordium is often observed in cases in which the heart disease began in childhood. The abdomen should be carefully inspected for abnormal pulsations, especially in the hepatic area. Capillary pulsations may be observed in the fingernails or lips and are suggestive of aortic regurgitation. Clubbing of the fingernails may be associated with congenital cardiac disease and is often seen in subacute bacterial endocarditis.

b. Palpation.—Palpation serves to confirm and amplify many of the findings of inspection. The following points should be especially well covered: apex beat, thrills, pulse, abdomen, and chest. The position, extent, and force of the apex beat should be determined. Here, we have a confirmatory indication of the size of the heart. Thrills are best determined by palpation. A thrill is discernible as a fine, tingling or purring sensation felt over the precordium, which bears a constant relationship to some phase of the cardiac cycle. In order to elicit a thrill, it is important that the examining band be placed lightly over the precordium. By slightly raising the palm of the hand and creating thus a small suction disk, thrills can often be detected much more easily. The radial pulses are always palpated to determine the character of the pulse, its force, volume, rhythm, and rate. Comparison of corresponding arteries in the same individual may show a retardation, difference in amplitude, or obliteration of one. The abdomen should be palpated to determine the presence of any abnormal pulsations, aortic, splenic, or hepatic. The size of the liver and spleen should also be checked and the amount of ascites determined. Abnormal pulsations of the chest may be found which were not discernible upon inspection.

c. Percussion.—(1) Percussion of the heart is of questionable value at the present time; it is extremely useful and accurate at the hands of certain men, of little or no value for the rest. About the only information that the average man can derive from this procedure is an indication of marked enlargement, marked displacement, or large effusions. The chief inaccuracies arise in technique, anatomical relationships, and interpretation. Variation in the force of percussion stroke and variation in the pressure of the exploring finger lead to differences in the elicited note which may easily be misinterpreted. The thickness of the chest wall, the amount of lung tissue lying between the heart and chest wall, and the movements of the heart itself within the chest also becloud the findings. And, finally, the subjective element of interpretation leads to wide variations. The accuracy with which X-ray determines enlargement, as well as

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determining type and degree, practically eliminates percussion, other than confirmatory, as a method of diagnosis.

(2) For the information of those who must report percussion findings, or who do not have access to an X-ray, the following normal standards are given: The left border of dullness is the most easily and most accurately percussed. In the fifth left interspace the left border measures 8 to 9 centimeters from the midsternal line. This is the maximum measurement and coincides within 1 centimeter to the PMI (dullness usually is a bit to the left). In the third left interspace the border of dullness measures half that of the fifth left interspace. The right border of cardiac dullness and that of the great vessels are percussed with extreme difficulty. The right border coincides with the right edge of the sternum. The great vessel dullness coincides with the sternum at the level of the first and second interspaces. Only in case of pathologic enlargement or shift in position of the heart will either the right border or the great vessels be percussed.

d. Auscultation.—(1) There are two types of auscultation practiced today, direct and indirect. Direct auscultation involves the use of the unaided ear directly over the area involved. Indirect auscultation interposes a stethoscope between the ear and the area to be evaluated. There are many types of stethoscopes, each with its proponents, and each of value to its user. It is a good policy for a beginner to select one type of stethoscope and use it exclusively until he has learned to evaluate the sounds he hears therewith. Many experienced cardiologists use both the bell type and Bowles type in the same examination, each one being suited to bring out certain groups of sound characteristics. In the past few years, amplifying stethoscopes and recording stethoscopes have come into use; but their value for the present time is limited to research and teaching purposes.

(2) In listening for heart sounds, the following areas should be thoroughly covered:

(*a*) Apex (fifth left interspace 9 centimeters from the midsternal line).

(*b*) Tricuspid area (left side of middle and lower sternum).

(*c*) Aortic area (second right interspace at the border of the sternum).

(*d*) Pulmonary area (second left interspace at the border of the sternum).

(*e*) Lungs (both apices and bases) for signs of congestion.

(*f*) Neck vessels for transmission of murmurs.

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Sounds at the base of the heart are best heard with the patient in the upright position, while those at the apices are best heard with the patient in the reclining position. To bring out distant sounds, one should have the patient exhale completely and hold this position momentarily, if possible. This act reduces the amount of air bearing tissue between the stethoscope and the heart and eliminates the distracting respiratory sounds. Often in this way otherwise obscure sounds, and especially murmurs, are clearly brought out. Likewise, small changes in position, either upright or reclining, which tend to throw the heart against the chest wall, will accomplish the same purpose and bring out or amplify distant or indefinite sounds.

(3) The normal heart has two distinct sounds with each beat, and at rare intervals, a third, which when present is heard with difficulty. The sounds are named first, second, and third, according to their place of occurrence in the cardiac cycle. The third sound is of no importance under normal conditions and will not be mentioned again except under the heading of gallop rhythm. The first sound is loudest over the apical areas and the second sound is best heard over the base. These sounds are classically described as flub-dub, the first or flub being low pitched and rumbling, while the second or dub is higher pitched, sharp, and snapping. Three characteristics of the sounds should be noted—quality, loudness, and rhythm. Quality is the most important ausculatory feature. It is a property of heart sounds that cannot be described and interpretation can only be acquired through experience. Listening to a thousand or more of the healthy young applicants for flying training gives a good conception of the quality of normal heart sounds. In no other way than by actual listening to many normals can this feature of cardiac judgment be gained. The loudness or force of the heart sounds varies widely with the condition of the muscle in youth, age, and disease; also, with the thickness of chest wall tissue to be listened through. In general, the louder the beat, the more forceful is the muscle contraction. The force of the second sounds at the base deserves special consideration. In youth and health the pulmonary second sound slightly exceeds the aortic second sound. In pulmonic diseases and congestion this is much more accentuated. In age, and especially in hypertension, the aortic second sound exceeds the pulmonary second sound, and often has a ringing or tambour quality. The third factor, rhythm, is best considered under the arrhythmias (pars. 190–195).

(4) Murmurs are of great importance and, contrary to the present popular opinion, are of such great importance that section II (pars.

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42 to 50) is being devoted to their detection and significance. It will be sufficient to say at this time that pitch, intensity, quality, timbre, point of maximum intensity, relationship to events in the cardiac cycle, and the direction of their transmission over precordium, back, and neck vessels, should be noted.

(5) The friction rubs of pericarditis constitute the final feature of auscultation. These are most commonly heard over the left edge of the sternum. They vary widely in timing and character. Usually, they are both systolic and diastolic, having a loud swish-swish character, but occasionally they are systolic alone. When systolic alone they can be differentiated only with difficulty from systolic murmurs. In character they vary from soft, blowing, to loud, rough, grating sounds. Another feature which adds to their difficulty of interpretation is their inconstancy and variability with shift of position. Friction rubs of any character mask heart sounds and murmurs and render diagnosis of heart conditions other than pericarditis rather difficult.

e. Estimation of cardiac reserve.—The history gives control findings in this procedure and it is to be again emphasized that this estimation has its greatest value when compared to predisease normals. The degree of failure or lessening of the cardiac reserve can be brought out either by a simple observation or a series of simple tests. Persons who have lost their reserve have obvious congestive failure. Persons with moderately diminished reserve are dyspnoeic after moderate amounts of ordinary activity, such as walking briskly on a level, up slight grades, or climbing stairs. Persons who have lost but a slight amount of reserve find that they cannot do their ordinary activities as strenuously as before, due both to fatigue and dyspnoea. This is especially true when the patient tries to speed up activity or hurry. For persons in this group one or two simple tests will suffice. Hopping on one foot 100 times, clearing the floor at least 1 inch with each hop, is a standard Army procedure. (Par 64, AR 40–105.) A normal response is an increase in pulse rate immediately after exercise not to exceed 50 percent of the preexercise normal, and a return of the pulse rate to nearly normal (usually within 8 beats) after 2 minutes' rest. This is a very simple and very satisfactory test and has the additional value of indicating lack of condition in normal hearts. Sir Thomas Lewis describes two good exercise tolerance estimation procedures, a simple test and a strenuous test. In the simple test the subject walks briskly up a flight of 40 steps, 1 step at a time. A person in good condition will show no respiratory effort and the pulse rate will not rise more than 10 or

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20 beats per minute. Dyspnoea of any degree or greater increase in pulse rate indicates some myocardial impairment. In the strenuous test the subject lifts a weight of from 10 to 20 pounds from the floor to above his head at the rate of 1 lift every 2 seconds. A normal person can accomplish this feat 30 to 60 times before breathlessness forces him to stop. This latter test obviously should not be given anyone suspected of having serious myocardial pathology, especially of the coronary type.

41. Blood pressure.—*a.* Arterial blood pressure is measured almost universally by the compression cuff method. The finger compression method used by some of the older physicians is notoriously inaccurate. Interpretation of the compression cuff system is made by four methods: palpatory, subjective, auscultatory, and oscillometric. The auscultatory method is the most widely accepted. Here, the first sound heard after releasing cuff pressure is taken as the systolic, and the point of disappearance of sound is taken as the diastolic pressure. In the palpatory method, systolic only can be determined, the point where the first pulse beat is felt to go through the radial artery being taken as the systolic blood pressure. This reading is usually 5 to 10 points (measured in millimeters of mercury) below that of the auscultatory method. The diastolic pressure cannot be secured by the palpatory method. In the subjective method, the subject himself interprets both diastolic and systolic pressures. The systolic pressure is taken as the point where the first arterial thrill is felt as the blood rushes into the empty artery; the diastolic pressure is that point where the thrill ceases. This method also gives readings 5 to 10 points below the auscultatory. The oscillometric method is the one used in most self-recording sphygmomanometers. This method is more accurate and more complex, requiring a double cuff and an elaborate piece of apparatus. There are several types of visually read oscillometric sphygmomanometers, somewhat simpler of operation, but having no real advantage over the auscultatory method. The oscillometric method is accepted generally as the most scientifically accurate.

b. The auscultatory method presents five distinct phases, as heard in the brachial artery as decompression of the cuff is carried out. The beginning of the first phase is heard as the first clear, ringing sound. The beginning of the second phase is heard as the point where the clear, ringing sound changes into a murmur with each pulsation of the arterial column. The beginning of the third phase is heard as this murmur again changes into a clear, ringing sound. The beginning of the fourth phase is heard as this clear, ringing sound changes into a dull thud. The beginning of the fifth phase is heard as the point where

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all sound ceases. The systolic pressure is taken as the beginning of the first phase, and the diastolic pressure is taken by most men as the beginning of the fifth phase, although many insurance companies and others indicate that the beginning of the fourth phase should be taken as the diastolic blood pressure. The duration of each phase is given as follows: first, 5 to 15 millimeters below systolic pressure; second, 10 to 20 millimeters below first; third, 15 to 25 millimeters below second; fourth, 5 to 10 millimeters below third to silence. In some cases considerable difficulty is experienced in identifying these phases, and in all cases practice is needed to be absolutely sure.

c. One error commonly made in using the auscultatory method is the misinterpretation of the auscultatory gap. The auscultatory gap is a period of silence occasionally occupying the second phase. It is heard especially in cases of hypertension, and when the cuff is applied too tightly or inflated for too long a period, or there is some other constriction to the circulation of the arm, such as a tight sleeve. When the gap is present, the first phase will be heard distinctly, and then there will be a period of silence for 20 or more millimeters, and then the third phase will be resumed. The possibilities of error lie in that the beginning of the third phase is often taken as the systolic pressure and, less frequently, the end of the first phase is recorded as the diastolic. These errors are more apt to occur when the spread of the phases is increased, as in hypertension.

d. Army Regulations prescribe that blood pressure will be taken by the auscultatory method. Either the mercury or the needle gage type of aneroid sphygmomanometer may be used, although the mercury type is to be preferred. Mercury type instruments should not be accepted if the inherent error is more than 3 millimeters per 300 millimeter column of mercury. The aneroid type of instrument should be checked frequently against a mercury manometer for accuracy.

e. Paragraph 65, AR 40–105, prescribes that, "No applicant will be rejected as a result of a single reading. When the blood pressure estimation at the first examination is regarded as abnormal, or in case of doubt, the procedure will be repeated twice daily (in the morning and in the afternoon) for a sufficient number of days to enable the examiner to arrive at a definite conclusion."

f. Paragraph 72*g*, AR 40–105, prescribes that, "Hypertension evidenced by a persistent systolic blood pressure above 150" is disqualifying. Also "In persons under 25 years of age a persistent systolic pressure above 140 is cause for rejection."

g. Paragraph 31*f*(1), AR 40–110 (pertaining to physical examination for flying, give the same standards as mentioned, plus the fol-

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lowing: "A systolic pressure less than 105 millimeters in itself disqualifies. A diastolic pressure 100 millimeters or over in itself disqualifies. In the case of applicants for flying training, a persistent systolic blood pressure of 135 millimeters or more, or a persistent diastolic blood pressure of 90 millimeters or more, or an unstable blood pressure disqualifies."

42. Recommended texts.

Heart Disease (especially first edition) (White)	Macmillan.
Diseases of the Heart (Lewis)	Macmillan.
Physical Diagnosis (Cabot)	William Wood and Co.
Diseases of the Chest and the Principles of Physical Diagnosis (Norris and Landis)	Saunders.
Diseases of the heart (Vaques and Laidlaw)	Saunders.
Heart Affections (Calvin Smith)	F. A. Davis Company.
Diseases of the Heart and Aorta (Hirschfelder)	Lippincott.
Clinical Features of Heart Disease (Crummer)	Hoeber.

Section II

CARDIAC MURMURS AND THEIR SIGNIFICANCE

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43. Cause.—Cardiac murmurs are abnormal sounds arising within the heart, due to abnormal conditions of the valvular or muscular structure. Mechanically, murmurs are caused by one of two conditions, the passage of the blood stream through a narrowed orifice into a larger cavity, or the flapping of a torn piece of tissue, valve, or muscle in the blood stream. The mere passage of fluid through a narrow opening alone will not produce a murmur. It is necessary that this fluid be under pressure and, upon leaving the opening, enter a much larger cavity. The suddenness of this entry (the jet action) sets up eddy currents. The murmur sound itself, in this case, is caused by vibra-

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tion of the muscle or vessel wall, as the result of the eddy current. When these vibrations are palpable, they are called thrills.

44. Detection.—*a.* Many murmurs, especially those due to the flapping of a piece of tissue in the blood stream, are loud and easily heard with the unaided ear. Others require special attention and procedures to bring them out. The same general rules that have been noted relative to heart sounds also apply to murmurs. Those at the base are best heard when the patient is in the upright position and those at the apices when the patient is reclining. Holding the breath in the expiratory position is helpful, and changes of position which throw the heart against the chest wall are of considerable value.

b. Perhaps the surest method of bringing out a latent murmur is to have the patient perform mild exercise if he is able. Hopping on one foot 100 times will bring out most organic murmurs. These will be easily heard if the patient is placed in the most favorable position immediately after the exercise. Most murmurs that require exercise to bring them out subside as soon as the heart returns to its normal rate. Exercise brings out murmurs by increasing the heart rate and flow of blood through the heart. The increased flow of blood through a narrowed opening increases the eddy currents, and thus increases the vibration of the vessel walls.

c. Proper use of the stethoscope will make detection simpler at all times. In obscure cases, both the bell and Bowles types should be used. Each has its value with sounds of different pitch, the Bowles type being most useful in sounds of higher pitch. Extrinsic stethoscope sounds are minimized by the use of heavy tubing which should be about 18 inches long. A thumb rest on the bell portion also reduces the possibilities of extraneous noises. Light pressure against the chest wall seems to bring out some types of murmurs, and it has been found that by simply allowing the weight of the stethoscope bell to hold itself in position against the skin creates the optimum conditions necessary to hear the fainter sounds. It is only rarely that pressure with the stethoscope bell will be of any assistance.

45. Characteristics.—In evaluating cardiac murmurs, all of the following characteristics must be considered:

Pitch.
Quality or timbre.
Intensity.
Point of maximum intensity.
Direction of transmission.
Relationship to events in the cardiac cycle.
Effect of extracardiac factors, position, exercise, and respiration.

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a. Pitch and quality have to do essentially with the musical characteristics of the sound. Pitch is easily classified into high, medium, and low. Quality or timbre of the sound is more difficult to describe and classify. The terms blowing, musical, harsh, rasping, rumbling, or grating fit the different types fairly well. Intensity or loudness falls easily into four grades, very loud, loud, moderate, and soft. The term crescendo is often useful in describing its special type of intensity. The point of maximum intensity localizes the murmur to one valve area, the PMI usually coinciding with a definite area of valve sound transmission when the degree of enlargement is taken into account. In general, the farther one gets from the PMI, the fainter the murmur.

b. Transmission of murmur sounds is the one exception to this rule, there being three murmurs which are transmitted so definitely that the area of transmission is one of the identifying features. Aortic systolic murmurs are characteristically transmitted into the neck vessels, very loud ones also being heard in the cardiac apex. Aortic diastolic murmurs are transmitted down along the left border of the sternum. Mitral systolic murmurs are transmitted into the left axilla and back. The mitral diastolic murmur characteristically has no transmission. In general, pulmonary and tricuspid murmurs have little or no transmission. This is due to the fact that the louder the murmur, the more extensive the area of transmission, and most murmurs in both pulmonary and tricuspid areas are inherently fainter than those on the left side of the heart.

c. The relationship of murmurs to events in the cardiac cycle falls under three headings, timing, length, and effect on sounds. Under timing, murmurs are either systolic, diastolic, or both. Murmurs may be of any length, occupying any portion of either systole or diastole. Likewise, they may be continuous with either the first or second sound, may replace either or both, or there may be a distinct interval between the heart sound and the murmur. In general, organic murmurs impinge upon or replace cardiac sounds.

d. The effect of the extracardiac factors, position, exercise, and respiration, have been touched upon previously. The value of position and exercise in bringing out or amplifying murmurs has been mentioned. Likewise, the effects of respiration utilized for the same purpose have been noted. One additional feature wherein respiration helps is that, in general, cardiorespiratory murmurs cease upon holding the breath and are amplified by rapid and deep breathing. Although this is not an absolute rule, it should be borne in mind.

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46. Significance.—Contrary to the present trend of thought among internists, cardiac murmurs play a great part in evaluating the disturbance of the cardiac economy. However, no diagnosis is satisfactory which rests upon the evidence of murmurs alone. Likewise, the seriousness of the disease is not indicated by the loudness or softness of the murmur. In fact, in many cases, as the condition becomes worse, the murmur becomes fainter and as improvement sets in the murmur becomes louder. Again, not all serious cardiac disease is manifested by murmurs. Many diseases, especially coronary types, degenerative myocardial disturbances, and even organic valvular diseases can and do exist in the absence of all murmurs. As to the individual murmurs in general, systolic murmurs are not in themselves indicative of organic disease, whereas diastolic murmurs are entirely of organic origin. In the absence of other signs of myocardial pathology, systolic murmurs are usually of no organic significance, and a diagnosis of disease should never be made on systolic murmurs alone. That is to say, on one hand, if there is a systolic murmur and no physical sign of heart disease, the murmur is to be ignored; and, on the other hand, if there is a systolic murmur and some other physical sign of heart disease, such as cardiac enlargement, then the diagnosis of organic heart disease is to be based on such signs and not on the systolic murmur only. Conversely, diastolic murmurs nearly always mean organic valvular disease and a diastolic murmur, even in the absence of other signs, is more than presumptive evidence of such. However, as previously stated, a diagnosis based on a diastolic murmur alone is not a satisfactory one, and a more thorough search for other findings to support the diagnosis is indicated.

47. Systolic murmurs.—The systolic murmurs are best discussed under five headings: cardiorespiratory, pulmonic, apical, aortic, and tricuspid.

a. The cardiorespiratory murmur is quite the most frequent. It is an extracardial sound originating from the rush of air into alveoli near the heart, which are decompressed with each systole. The point of maximum intensity of this murmur is usually at the base, although it may be heard over a wide area of the precordium, and even at the angle of the left scapula. It is readily recognized, being a short, rather superficial whiff, which is audible only (or most audible) during inspiration. It is systolic in time. Holding the breath abolishes this murmur and readily makes the differentiation. The murmur has no diagnostic or prognostic significance. It is usually associated with rapid heart action.

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b. The pulmonary systolic murmur is the next most common. It is a systolic murmur, soft or harsh, audible over the second, third, or fourth left costal cartilages. It is often heard only when the patient lies down. Exercise abolishes it in most instances. It has no significance, but may be used in diagnosis to remind the examiner that the heart's base should be palpated for thrills. Neither cardiorespiratory nor pulmonary murmurs impinge on the heart sounds, and neither have a true transmission.

c. The apical systolic murmurs are of three types: a functional murmur audible only when the patient is in one posture (standing or lying, usually the latter), the murmur of relative mitral insufficiency, and the murmur of organic mitral regurgitation. The systolic murmur at the apex is usually soft, blowing, high pitched. When it is organic, it is continuous with or replaces the first sound and is transmitted into the left axilla or to the angle of the left scapula. However, when one assumes that this murmur is organic, the diagnosis of mitral regurgitation is uncertain. The differentiation between systolic murmurs due to a lax ring (relative mitral insufficiency) and those due to a damaged valve is not possible from the character of the murmur itself. The only sure indication of disease of the mitral valve is the diastolic, apical murmur of mitral stenosis. However, in children with a history of one or more of the rheumatic manifestations, the diagnosis of mitral valve disease can generally be made in the presence of cardiac enlargement and the systolic murmur. The diagnosis, in this case, is based as much on the history and the cardiac enlargement as on the murmur itself.

d. The aortic systolic murmur is less frequent than the pulmonary systolic murmur and, in young men, has no greater significance. This murmur is harsh, rough, loud, impinges upon or replaces the first sound, and is transmitted into the vessels of the neck. It is caused by three conditions: aortic stenosis, dilatation of the aorta as in aortitis, and the dilatation of aortic aneurysm. Aortitis is far the most frequent of the three as a causative agent. The murmur is not to be taken as a sign of aortic stenosis, in that almost all cases of stenosis present a systolic murmur but, conversely, very few cases which present an aortic systolic murmur are cases of stenosis.

e. The tricuspid systolic murmur, similar to the mitral in characteristics, is heard at its maximum intensity over the lower part of the sternum. It is usually associated with tricuspid regurgitation. The tricuspid valve is more easily rendered incompetent than is the mitral. Strenuous exercise is sufficient to produce the murmur over the ensiform cartilage in many healthy people. This murmur is an incident

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which may be associated with clear signs of heart failure, but it is also an incident which happens in perfect health. The value of the tricuspid systolic murmur in prognosis is therefore negligible.

48. Diastolic murmurs.—The only murmurs of importance in the diagnosis of valvular heart disease are the diastolic murmurs. They are less frequent in occurrence than systolic murmurs and often harder to hear. Diastolic murmurs heard in either basal area have very similar characteristics and it is difficult to distinguish between them. The same fact holds true for diastolic murmurs in either apical area. In general, basal diastolic murmurs usually indicate aortic regurgitation and apical diastolic murmurs (including presystolic murmurs) usually indicate mitral stenosis. However, for the sake of clarity, each area will be considered individually.

a. At the apex or mitral area two murmurs are heard; that of mitral stenosis and that of aortic insufficiency. The murmur of mitral stenosis is low, rumbling, often described as crescendo, heard only at the apex, and has no transmission area. It occurs late in diastole, in the presystolic period, and is usually continuous with and ends in the first heart sound. It is heard best with the patient lying down, and some authors state it is best heard with the bell type stethoscope. The louder murmurs are frequently accompanied by a thrill. Often when the patient is in the upright position the murmur will not be heard and its presence will be indicated only by an accentuated first sound in the mitral area. When this condition exists and one suspects the presence of a murmur from the accentuated or roughened first sound, exercise and postural changes will bring out this murmur. The mitral diastolic murmur of aortic regurgitation is known as the Austin Flint murmur. It is distinguished from the murmur of organic stenosis by the fact that it is a softer murmur, occurs earlier in diastole, being more apt to follow closely the second sound than to be continuous with the first, and that it is never accompanied by a thrill.

b. At the aortic area, the only diastolic murmur of any importance is that of aortic regurgitation. This murmur is soft, blowing, high pitched, usually occupying all of diastole. It either is continuous with the second aortic sound, impinges upon, or replaces it; in fact the louder varieties often mask both the first and second sounds. It is best heard with the patient in the upright position, and as some authorities state, with the Bowles type stethoscope. There is no thrill. The area of transmission is down the right side of the sternum, loudest in the middle portion.

c. The diastolic murmurs occurring on the right side of the heart have essentially the same characteristics and causation as those occur-

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ring on the left. In the tricuspid area, the diastolic murmur indicates tricuspid stenosis, a diagnosis rarely made. The murmur is somewhat more musical and much softer than that of mitral stenosis and is heard over a wide area at the base of the sternum. In the pulmonary area, the diastolic murmur indicates pulmonary regurgitation, a very rare condition. Most murmurs heard in the pulmonary areas have their origin and PMI in the aortic area. However, when pulmonary regurgitation does exist, the murmur is identical with that of aortic regurgitation, except that the PMI is at the pulmonary area, and its area of transmission is down the left side of the sternum. This murmur is called Graham Steelle, when mitral stenosis is the cause of the pulmonary regurgitation.

49. Functional murmurs.—*a.* Prior to this point, almost everything that has been said about murmurs has referred to the organic type. However, functional murmurs are perhaps more numerous and have a more variable sound pattern. The difference between functional and organic murmurs is best defined thus: A functional murmur is one occurring within the heart in the absence of any valvular disease; and an organic murmur is one occurring as the result of valvular disease or structural defect.

b. In differentiating functional from organic murmurs, the following points should prove helpful. Functional murmurs—

(1) Are nearly always systolic in time.

(2) Have no transmission.

(3) Do not replace or impinge upon heart sounds.

(4) Are usually soft, blowing in type.

(5) Are usually of short duration, being loudest at the end of systole.

(6) Are very variable in character, exercise or deep breathing abolishing them momentarily.

(7) Occur most frequently at the pulmonary valve area, although it is to be remembered that they often do occur at the mitral valve area.

While all of the above points are helpful in distinguishing between organic and functional murmurs, it should be remembered that the cardinal distinguishing factor is the presence or absence of other signs of organic heart disease. In the presence of other indisputable signs of organic disease, the above characteristics have little or no value, although in the absence of such signs, they constitute the distinguishing features.

50. Army Regulations.—Paragraphs 68, 69, 70, 71, AR 40–105, are entirely occupied with the differentiation of murmurs. Para-

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graph 72 states that any valvular disease of the heart is disqualifying for enlistment. Paragraph 73 states, "No candidate with a systolic murmur at the apex which is transmitted to axilla or angle of scapula will be accepted for commission."

51. Recommended texts.

Heart Disease (White)	Macmillan.
Physical Diagnosis (Cabot)	William Wood and Co.
Diseases of the Heart (McKenzie)	Oxford Medical Publications.
Diseases of the Chest and the Principles of Physical Diagnosis (Norris and Landis)	Saunders.

Section III

CHRONIC VALVULAR HEART DISEASE

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52. General.—*a.* Valvular heart disease is of extreme importance to aviation examiners in that any valvular abnormality, no matter how slight, is always a weak point in the individual's physical make-up. This weak point is subject always to further damage in either acute infections or severe physical strain. Army Regulations recognize this fact and state that "all valvular diseases of the heart" are cause for rejection. Minor degrees of valvular damage may escape detection at physical examination due to lack of presenting signs and symptoms. While such missed lesions are apt to cause little trouble, those which do cause cardiac disability in the future will constitute the bulk of that group of heart cases listed as of puzzling or unexplained etiology.

b. The causes of valvular heart disease are, in order of frequency: infection, arteriosclerosis, congenital lesions, and trauma. The infections are rheumatic fever, syphilis, tuberculosis, acute endocarditis, subacute bacterial endocarditis, and that terminal type of endocarditis which accompanies some of the acute infectious diseases. Rheumatic fever and syphilis are of the most importance in that they cause severe valvular damage which ultimately results in varying degrees of cardiac invalidism. Tuberculosis is relatively unimportant in that it rarely causes valvular damage. The endocarditis group is of no importance

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to examiners in that the diseases themselves are fatal and the valvular involvement is only an incident in the course of the disease rather than an end result. Arteriosclerosis, which ranks next to infections in importance, causes valvular damage by thickening, fibrosis, and calcification of the valve leaflets. Congenital lesions are usually anomalies in the number of leaflets in a single valve or such deformities as atresia, constriction of valve rings, or defective leaflets. Trauma is a very rare causative factor, and both trauma and the congenital lesions may be disregarded.

c. All of the major valves of the heart are subject to pathological damage. The mitral valve is involved most frequently. It is damaged in more than half of all cases of valvular disease. The aortic valve is the next most frequently affected. The tricuspid valve is involved rather infrequently and the pulmonary, rarely. Quite often more than one valve is damaged, two or more being involved in the same process simultaneously. This is best illustrated by the coincident involvement of both the mitral and aortic valves in rheumatic heart disease. The simultaneous involvement of the aortic, mitral, and tricuspid valves by the same disease is not uncommon. All four valves are involved together in approximately 1 percent of valvular heart disease.

d. Chronic valvular damage consists of one or both of two conditions, stenosis and incompetence. More frequently than not, they occur together in the same valve, the incompetence usually being the forerunner. Stenosis is always the end result of damage, usually inflammatory to the valve leaflet or cusp itself. Incompetence may be due either to actual disease of the cusps, as in rheumatic fever, to a dilatation of the valve ring, or to a generalized cardiac dilatation which involves all of the rings. Therefore, incompetence can be either organic, relative (due to stretching of the ring), or both. It is very difficult to differentiate between relative and organic incompetence prior to autopsy.

53. Mitral.—*a.* The chief and for all practical purposes the only cause of mitral valvular disease is rheumatic infection. Syphilis, tuberculosis, and congenital lesions are very rare causes. The fatal infections mentioned above attack the mitral valve very frequently but, as noted, are of little importance in this work. Therefore, being chiefly a rheumatic end result, mitral disease is an affliction of the young and middle aged, those falling in the enlistment group. Sex incidence is predominantly female, some authors giving figures showing mitral disease in women occurring twice as often as in men.

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b. It is better to think of mitral valvular involvement as "mitral disease" rather than as either mitral stenosis or mitral regurgitation alone. In the pathology of rheumatic involvement of this valve, incompetence precedes stenosis in all cases. The scarring which results in healing of the rheumatic valvular inflammation causes first an incompetence, then stenosis, which occurs rather late in the process. The heart is able to take care of most degrees of mitral incompetence by the process of hypertrophy and dilatation, and thus accommodate itself to that condition. However, once the process has become serious enough to be definitely stenotic, and thus cause incomplete opening of the valve, the cardiac reaction is a vicious cycle, and there is from that point on a definite progression toward cardiac termination. In other words, the milder stages of incompetence caused by scarring of the valve margins can be handled with some degree of ease by the cardiac reserve; but the resistance of the fixed stenotic valves creates a load which the heart is unable to overcome, and so gradually fails in trying.

c. In mitral disease the first insult to the valvular autonomy is a special type of inflammatory reaction along the line of apposition of the cusps (not the valve edges) known as vegetations. These vegetations are tiny fibrous excrescences and occur in rows on the line of contact. The inflammatory reaction spreads from the vegetations into the cusps themselves and often onto the neighboring endocardial wall. When the active inflammatory process subsides, healing takes place by the formation of scar tissue. The normal contraction of this scar tissue causes deformities along the apposition edges of the valve leaflets, thus preventing the valve from closing tightly. This results in the early stages of incompetence. As the valve is attacked again and again by the rheumatic inflammation, each incident adds new inflammatory products, and as an end result, new scar tissue. Thus, as each new batch of scar tissue contracts in healing, it adds to the deformity of the valve, increasing the incompetence. When enough scar tissue has been laid down so that the valve edges are thick fibrous masses, stenosis is present due to the immobility of the now fibrous valve cusps. An additional factor in the production of incompetence is inflammation of the chordae tendineae. These shorten in the contraction process of healing, and thus, by their shortening, aid in preventing the valve leaflets from closing tightly. An additional factor in the production of stenosis is the sealing together of valve leaflet edges at their bases near the ring. This adherence is likewise an end result of inflammatory healing and reduces considerably the opening through which blood can flow during auricular systole and adds to the burden created by

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the stiffened valve leaflets. The end result of mitral disease is the "buttonhole" valve, a rigid, fibrous, valvular opening, considerably reduced in size, which has little or no activity in the cardiac cycle.

d. Mitral regurgitation caused by incompetence of the valve is, therefore, a rather uncertain and a rather transient diagnosis. There are no symptoms which cannot be traced to the underlying disease or the end results of the valvular disease. Two signs are necessary to make an absolute diagnosis, the characteristic murmur and cardiac enlargement. The murmur is constant, high pitched, blowing, usually soft, heard best at the cardiac apex, transmitted into the axilla, systolic in time, continuous with or replacing the mitral first sound, heard best in the prone position, increased by exercise and brought out by holding the breath in expiration. The cardiac enlargement may be generalized, but the left auricle specifically will be enlarged. As previously mentioned, regurgitation exists more often in conjunction with stenosis and nearly always is a predecessor to stenosis when it exists alone.

e. Mitral stenosis, like regurgitation, has no characteristic symptoms. The diagnosis cannot be made in the absence of the characteristic murmur. Other signs are present, but are only suggestive when the murmur is absent. The murmur is low pitched, rumbling, crescendo, best heard at the cardiac apex, has no transmission, is mesodiastolic or presystolic in time and ends in the first mitral sound. Often it is not heard at all in the upright position. Exercise brings this murmur out very well, although one may have to listen quickly at the apex to hear it, for it often disappears before the tenth beat of the heart after exercise. If one has the patient in the prone position on the left side listening within the first ten or fifteen beats following exercise and then does not hear the characteristic murmur, he is safe in saying that the murmur does not exist. There are three accompanying signs which, when heard, should suggest to the listener that there might be mitral stenosis present and that effort should be made to bring out the characteristic murmur. These signs are: roughening, accentuation or reduplication of the mitral first sound; accentuation of the pulmonary second sound; and a mitral systolic murmur. These are of utmost importance to a routine examiner in that the murmur of mitral stenosis is not always apparent and must often be searched for. Other signs besides the murmur to be noted are a precordial thrill, left auricular enlargement, congestion of the lungs, and right ventricular enlargement. These are all (except the thrill) secondary results to the degree of effort expended by the heart in overcoming the added burden

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imposed by the stenosis. Terminal end results of mitral disease are congestive heart failure and auricular fibrillation. Persons with mitral disease have a definitely poor prognosis and are especially subject to intercurrent infection. Most die before the age of fifty either of congestive failure or some intercurrent condition.

f. Two other very much less common causes of mitral valvular disease should be mentioned in passing, sclerosis and dilatation. Arteriosclerosis of the valve leaflets occurs as one of two processes. There may be an arteriosclerotic infiltration of the aortic cusp of the mitral valve due to extension of the process from the neighboring aorta, or calcium may be independently laid down upon areas of the valve previously damaged by rheumatic infection. Both of these conditions are causes of stenosis not usually preceded by insufficiency. The other condition of importance is chronic generalized dilatation of the heart, in which the mitral valve ring enlarges along with the left ventricle. This causes relative incompetence of the valve due to stretching of the two leaflets and their chordae tendineae. Here, the leaflets also spread apart, causing the interspaces to enlarge. This results in incomplete closure of the valve at the cusp base, further adding to the incompetence.

54. Aortic.—*a.* Aortic valvular disease is caused in the greater majority of cases by one of three conditions: syphilis, arteriosclerosis, and the rheumatic infections. Syphilis is by far the most important in the south, especially among the Negro population. Arteriosclerosis is of much wider distribution and general importance. Rheumatic fever ranks a poor third. Of the minor causes, such conditions as hypertrophy and dilatation, the fatal types of endocarditis, congenital abnormalities, and trauma should be noted. Hypertrophy and dilatation are rather rarely the cause, usually the result, of valvular disease. The early fatal termination common in endocarditis has been previously mentioned as eliminating this group from the consideration of chronic aortic valvular disease. Congenital abnormalities are rare and when they do occur are usually anomalies in the number or shape of the valve cusps. Trauma, also rare, usually causes rupture of an aortic cusp at the site of previous damage. Rupture of a leaflet causes an immediate and severe aortic insufficiency, which in turn causes death in a short time or, if survived, is distinguished by its severity and a tremendously loud murmur. Aortic valvular disease is found in men three times as frequently as in women. Of the three principal causes, rheumatic involvement is a disease of youth, syphilitic involvement a disease of middle age, and arteriosclerotic involvement a disease of old age.

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b. The pathology of aortic valvular disease differs considerably from that of the mitral valve, although they have some common characteristics. In the mitral valve, stenosis is always preceded by incompetence. In the aortic valve, this may also be true, but stenosis does exist without incompetence, and insufficiency of fatal termination frequently exists without ever reaching the stage of stenosis. Arteriosclerotic infiltration of valve cusps illustrates pure stenosis and syphilitic insufficiency illustrates pure insufficiency. The insufficiency exists alone much more frequently than does the stenosis. Syphilis causes incompetence of the aortic valve by a stretching of the valve ring secondary to involvement of the aorta. Arteriosclerosis accomplishes incompetence in the same manner, the stretching of the valve ring being secondary to dilatation of the aorta in atherosclerotic aortitis, especially when the aortitis is accompanied by hypertension. Cardiac hypertrophy or dilatation, especially of the left ventricle, when it is the cause rather than the effect, also stretches the valve ring. When the ring is stretched, the bases of the leaflets are pulled apart at their commissures and the apposition edges of the leaflets are tautened. This combination of circumstances prevents tight closure of the leaflets at any point on their contact margins and incompetence is the result. In view of the fact that the aortic ring is more severely involved and commonly more widely stretched than the other valves, it is easily seen why incompetence here is of such a serious import. Arteriosclerosis, besides stretching the valve ring, also can cause a pure stenosis by infiltration of the valve leaflets from the base, the process extending from base to free margin with an end result of fibrosis or calcification. Rheumatic fever produces the same type of pathology as seen in the mitral valve, going through the stages of inflammatory hyperemia, vegetations along the line of closure, scar tissue healing, contraction, and deformity. The first results are incompetence, the end result, stenosis. In rheumatic fever, the aortic valve alone is attacked in 5 percent of cases, and involved together with the mitral valve in over 30 percent. There is no distinctive progression in the pathology of the aortic valve as in the mitral valve which would justify grouping the lesions as one disease, so both aortic insufficiency and aortic stenosis must be considered individually to get the correct picture.

c. Aortic insufficiency is by far the most important and frequent of the two. It is of all degrees from the very slight, which easily escapes detection, to the very severe. Here, the aortic valve is no longer able to stop the backflow of blood from the systemic arteries during diastole, and blood rushes back into the left ventricle. This

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extra amount of blood to be pumped greatly increases the amount of work for the left ventricle, and the response of the heart to this added burden is a tremendous hypertrophy and dilatation. The largest hearts on record are seen in this condition, the enlargement in advanced degrees being so great as to warrant the application of the term "beef heart" or "cor bovinum." The other systemic effect of the incompetency is an increase in the pulse pressure. Inasmuch as there is little or no resistance to the backward thrust of the aortic column of blood, diastolic pressure is considerably reduced and a diastolic pressure of zero is not uncommon. This, coupled with the hypertension incident to the increased expulsive effort of the ventricle, gives a very wide pulse pressure.

d. The symptoms of aortic insufficiency are anginal pain and palpitation. The pain frequently may be severe and is most often aggravated by effort. It is caused by a combination of two factors, the diminution of the coronary circulation secondary to the lowered diastolic pressure (diastolic pressure determines the adequacy of the coronary circulation) and the increased demand for nourishment occasioned by the hypertrophy and increased work of the heart. The palpitation is due to the extreme extra effort of the heart in attempting to meet its increased load.

e. The diagnosis of aortic insufficiency can be made only in the presence of the characteristic murmur. This murmur is a high-pitched, blowing, rather faint sound, heard best over the second right interspace at the sternal border, transmitted down the sternum, especially to the third and fourth left interspaces. It is diastolic in time, occupies all of diastole, replaces the aortic second sound, and is frequently better heard with the unaided ear or a Bowles stethoscope. It is best heard with the patient in the upright position, leaning slightly forward, and with the breath held in expiration. Exercise increases the intensity, although at times a slow rate creates more favorable circumstances for hearing it. There are three related signs which should be present in addition to the murmur to make an absolute diagnosis: cardiac enlargement, especially of the left ventricle, Corrigan pulse, and a wide pulse pressure. Enlargement has been commented upon. The Corrigan pulse is a thrusting, rapidly rising and falling pulse which slaps the palpating finger and rapidly falls away. Capillary pulsations can often be seen by placing the fingernails under slight tension so as to blanche an area, or by placing a cover glass with slight pressure over the mucous surface of the everted lower lip. The low diastolic pressure with its resultant wide pulse pressure has already been mentioned. Another

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and lesser sign is the Austin Flint murmur, a diastolic murmur heard at the mitral area, due to vibrations of the mitral leaflets caught between the pressures of the two columns of blood flowing into the ventricle.

f. Aortic stenosis is much rarer than insufficiency. It sometimes exists alone, but in a far greater majority of cases occurs in conjunction with aortic insufficiency. It exists in all degrees from very slight to very severe. Here, the valve offers obstruction to the flow of blood from the left ventricle in systole. The result is simple hypertrophy of the musculature. In pure stenosis the left ventricle is hypertrophied only, whereas in insufficiency it is both hypertrophied and dilated. The dilatation of insufficiency is due to the larger volume of blood to be pumped by the heart as a result of the backflow. In pure stenosis there is no backflow, hence no dilatation, merely hypertrophy. Also, in pure stenosis, the diastolic pressure is maintained and the pulse is of the "plateau type," a slowly rising sustained pulse, with a flattened peak, rather than a thrusting pulse as in insufficiency. The causes of stenosis are two in number, calcification of the valve leaflet in arteriosclerosis, and the scarring fibrosis and fixation of the valve edges in rheumatic involvement.

g. Aortic stenosis produces no symptoms. Two signs are necessary to make the diagnosis, the characteristic murmur and its accompanying thrill. The murmur is low pitched, harsh, loud, located at the aortic area, and transmitted into the neck vessels. It is systolic in time, occupies all of systole, best heard in the upright position with the breath held in expiration, and is markedly accentuated by exercise. The thrill is limited to the aortic area and frequently is very intense. Related signs are a diminished or absent second aortic sound and the "plateau pulse" already mentioned.

h. The examiner should be very careful about making a diagnosis of aortic stenosis alone. It is a relatively rare condition and its murmur is intimated by two conditions in the aorta, aneurysm and arteriosclerotic aortitis. If the two required signs are present, and the imitating conditions ruled out, the diagnosis is plausible. However, the diagnosis is better made only in the presence of an accompanying aortic insufficiency, unless the examiner is absolutely certain of his data.

55. Tricuspid.—*a.* Disease of the tricuspid valve is essentially of no clinical importance. White states that in the first place deformity of the valve sufficient to be of clinical significance is rare. Secondly, when such deformity is present, it is usually overshadowed

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by a higher and much more important degree of mitral valve disease And thirdly, except in rare instances, tricuspid valve disease is not diagnosable ante mortem, though it may sometimes be suspected.

b. The causes of tricuspid valve disease are the same as the causes of mitral disease. Rheumatic fever is by far the predominating cause. In fact, there is an unusual parallelism between the tricuspid and mitral valvular involvements, differing only in degree. When the two are involved simultaneously, as they are in roughly 90 percent of cases of tricuspid disease, the mitral disease is usually so much more severe that the mere extension of the mitral signs will mask the tricuspid signs; thus, the tricuspid lesion is apt to be entirely overlooked. The pathology also is essentially the same as that of the mitral valve. The single exception to this is that relative dilatation is somewhat more frequent, due to involvement of the valve ring, in any venous congestion of more than moderate degree.

c. Tricuspid insufficiency, as noted, is caused by either organic valve disease or stretching of the valve ring, the latter being somewhat the more frequent. Organic disease is rheumatic in type, and the relative dilatation is associated either as cause or effect with venous congestion. Signs to be noted are the murmur and special evidences of venous congestion. The murmur is in every respect similar to the mitral systolic murmur of insufficiency except that it is located over the lower part of the sternum. The special signs of venous congestion accompanying this condition are marked pulsation of the right auricle and superior vena cava under fluoroscopy, liver pulsation, and sometimes pulsation of the spleen. Jugular pulsations can often be noted.

d. Tricuspid stenosis likewise presents signs very similar to mitral stenosis, and it is rather rare that a differential diagnosis can be made. The murmur again is the same, except that it is located over the lower portion of the sternum, and the related signs are those noted for tricuspid insufficiency.

56. Pulmonic.—*a.* Disease of the pulmonic valve is better defined and considerably less involved than disease of the tricuspid area. It is more frequently found alone with the other valves relatively normal than in combination with other valve lesions. Stenosis is only of congenital origin although rheumatic fever and the fatal types of endocarditis do attack it. When of congenital origin, the condition will give little trouble to a routine examiner, most stenosis being either a part of the more serious congenital conditions, such as tetralogy of

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Fallot, or so mild as to be missed entirely. Rheumatic involvement of the valve, although frequently very acute, has the rather unusual characteristic of leaving little or no residual damage. The involvement in endocarditis is merely an event in the fatal progression of the disease. Regurgitation is a relative process, being a stretching of the pulmonic ring secondary to the dilatation of the pulmonic artery in conditions which raise the pressure in the pulmonary circulation. Mitral disease and chronic lung conditions are the two greatest offenders.

b. Pulmonary stenosis gives no symptoms in itself, all symptomatology being secondary to the congenital combinations in which it occurs. The murmur and thrill are the two signs necessary to make the diagnosis. The murmur has all of the characteristics of the murmur of aortic stenosis, except that the PMI is in the second left interspace and there is no transmission to the neck vessels. The thrill is located over the PMI. The only related sign of any consequence in pure pulmonic stenosis is the resultant enlargement of the right ventricle. This is most frequently detectable only by means of roentgenology. To be absolutely sure of a diagnosis, aortic stenosis, aortitis, and aneursym must be ruled out.

c. Pulmonary regurgitation gives no symptoms in itself until so severe that right ventricular failure takes place. Right ventricular failure is much more common in regurgitation than in stenosis. The two signs necessary to make the diagnosis are the murmur and pulsation of the pulmonary artery and infundibulum. The murmur is in all respects similar to the murmur of aortic regurgitation and, from the standpoint of the murmur alone, cannot be distinguished from it. The PMI is in the second left interspace instead of in the second right interspace as in aortic regurgitation, but the transmission is down the sternum. If the Corrigan pulse is lacking, and there is marked pulmonary artery pulsation as seen either fluoroscopically or as a pulsation of the second left interspace, the diagnosis is fairly certain. The related sign of right ventricular enlargement is confirmatory to the diagnosis.

57. Recommended texts.

Heart Disease (White)	Macmillan.
Diseases of the Heart (Lewis)	Macmillan.
Clinical Heart Disease (Levine)	Saunders.
Diseases of the Chest and the Principles of Physical Diagnosis (Norris and Landis)	Saunders.
Physical Diagnosis (Cabot)	William Wood and Co.

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SECTION IV

CARDIAC ENLARGEMENT

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58. Types.—*a.* Cardiac enlargement is of two types, dilatation and hypertrophy. In dilatation the muscle stretches and the capacity of the various chambers increases. In hypertrophy the muscle itself increases in volume, but the capacity of the chambers remains unaltered. During life it is impossible to distinguish between hypertrophy and dilatation and only at autopsy can this be done by actually weighing the heart itself. In fact, in most cases, hypertrophy and dilatation coexist and it is rather rare that one condition be present for a very long period without the other developing.

b. It should also be remembered that the size of the heart varies with the size of the individual and the amount of physical activity indulged in. A 200-pound ditch digger will quite normally have a larger heart than a 100-pound clerk. The average weight of the adult male heart is 300 grams, and the average weight of the adult female heart is 250 grams.

59. Causes.—*a.* Dilatation is of two types, localized and generalized. The localized type is due to fibrosis of a portion of the heart wall, which area bulges slightly under the intercardiac pressure. Generalized dilatation is due to one of three types of causative conditions: inefficient beating of the ventricles, toxemias of the muscle, and inadequate blood supply to the muscle. Inefficient beating of the ventricles and the resultant lack of complete discharge of the ventricular contents are caused by the rapid ventricular rates seen in such conditions as auricular flutter and fibrillation. The relaxation of the muscle wall due to toxemias is found in pneumonia, diphtheria, scarlet fever, rheumatic fever, and the septic infections. The relaxation of the muscle wall due to inadequate nourishment is found in the conditions cutting down the coronary circulation and in the anemias.

b. It should be remembered that most of the conditions mentioned above are usually temporary and that when the causative condition

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is removed, the heart, unless too severely damaged, returns to its normal size and tone. Likewise, the pathological dilatations noted should not be confused with the slight physiological dilatation previously noted, by which the heart muscle increases its stroke volume.

c. Hypertrophy is the response of the heart muscle to an increased work load of a more or less permanent nature. It also is of two types, one, localized ventricular or auricular, and the other, generalized. The localized hypertrophy usually precedes the generalized hypertrophy and both are due to essentially the same causes, one being but a more serious degree of the other. The causes of the increase of the heart's work are persistent hypertension, valvular defects, and pericardial adhesions. The effects of the different lesions upon the individual ventricles are discussed herein, but it should be noted at this time that the resultant hypertrophy in mitral stenosis occurs in the left auricle and right ventricle and that the hypertrophy of aortic insufficiency occurs in the left ventricle. Arterial hypertension, likewise, chiefly affects the left ventricle, although the hypertrophy in this condition becomes generalized. The largest hearts seen at autopsy are those of long standing aortic insufficiency, the "cor bovinum."

60. Detection.—*a.* Cardiac enlargement displays no symptoms that could not otherwise be ascribed to either the underlying or the resultant conditions. However, the examination reveals three very definite signs, displacement of the PMI, X-ray findings, and electrocardiographic evidences. These signs are those of cardiac enlargement as seen in a normally placed heart; malposed hearts being very difficult to evaluate in size by any of the procedures. In malposition X-ray is the most helpful, the others being in most cases misleading.

b. In cardiac enlargement the borders of the heart extend beyond the normal limits of surface markings. The right border tends to appear beyond the edge of the sternum and the left border gradually moves into the axilla. Percussion is of little value, even in hypertrophy, due to its inherent inaccuracies, although some general ideas as to size may be gained by this procedure. The most reliable index to enlargement is the PMI as seen at the apex. Normally, this is seen in the fifth left interspace, 8 to 10 centimeters from the midsternal line. An impulse seen definitely beyond 10 centimeters or in the sixth interspace indicates enlargement. Likewise, a definite movement or thrust of bony structures over the PMI indicates enlargement. In normal hearts the PMI is seen as

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a movement of interspaces only, so that any beat so powerful as to give a thrusting impulse to the overlying ribs is to be considered pathological.

c. X-ray is the most accurate method of detecting cardiac enlargement and will always confirm the position of the PMI. Plates should be 6-foot flash exposures. Enlargement is definite when the maximum width of the heart is greater than half the width of the chest. Individual enlargement of the various chambers can be determined by either a flat plate or fluoroscopy. The roentgenogram is by far the most accurate method of determining hypertrophy of a single cardiac chamber.

d. Electrocardiographic findings are confirmatory evidence to other signs of enlargement only when the heart is not in malposition. Left axis deviation broadly indicates enlargement of the left ventricle and right axis deviation broadly indicates enlargement of the right ventricle. The degree of enlargement cannot be accurately estimated from electrocardiographic tracings alone.

61. Degrees.—*a.* Degrees of enlargement are best expressed according to the criteria set up by Sir Thomas Lewis. Three terms are used, considerable enlargement, moderate enlargement, and definite enlargement. In considerable enlargement the PMI and percussion border are far into the left axilla and there is forcible movement of a great portion of the chest wall. In moderate enlargement the PMI and percussion border are near the anterior axillary line, well down into the sixth interspace and there is some heaving of precordium. In definite enlargement the PMI is greater than 10 centimeters distance from the midsternal line, the percussion border perhaps detectable as being slightly to the left and there may be a definite thrust localized over the PMI. X-ray measurements are generally in accordance with the physical findings. As noted, degree of enlargement is difficult to determine by electrocardiograph.

b. It should be noted here that there are three types of normal heart positions and that two of these will sometimes be confused with smaller degrees of enlargement by the inexperienced. The three types are: the long heart, seen in asthenic individuals; the normal heart, with the normal oblique position; and the transverse heart, seen in stocky individuals with a high diaphragm. The long heart is centrally placed more or less in the long axis of the body and its PMI will frequently be in the sixth left interspace, although well within the normal 10 centimeters limit. The transverse heart is

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more or less transversely placed and its PMI will frequently be seen in the fourth left interspace just at or beyond the 10 centimeters limit. These two types should be borne in mind and the possibility of declaring one of these otherwise normal hearts enlarged should be avoided.

62. Significance.—Cardiac enlargement always means the presence of some pathological condition within the heart. There is no exception to this rule. Enlargement of a permanent character means that the heart muscle is weakened, less efficient, has lost some of its reserve and is more subject to such conditions as congestive failure. Given two heart cases with identical signs and symptoms except that one has considerably more enlargement than the other, the prognosis will be much worse in the case with the enlargement. In dilatation of an acute nature, in which the heart returns to normal size after the disappearance of the cause, there may be no detectable signs of damage, but it should always be considered that there has been some permanent residual damage, even though it may never manifest itself during the lifetime of the individual. It is a fairly authentic rule that hearts do not acutely dilate unless there is some weakness in the muscle prior to the disease. The mere fact that the pericardium is very difficult to stretch suddenly gives this hypothesis considerable weight. There is no treatment for cardiac enlargement except that of the underlying condition and the establishment of a regime that will enable the patient to live within the limits of his cardiovascular reserve.

63. Army Regulations.—Paragraph 67, AR 40–105, states "An apex beat located at or beyond the left nipple line, or below the sixth rib, and of heaving character, indicates an enlargement sufficient to disqualify for military service. Its cause, either valvular disease or hypertension in the majority of cases, should be sought for. Enlargement should not be made a primary diagnosis unless careful examination fails to reveal a cause."

64. Recommended texts.

Diseases of the Heart (Lewis)	Macmillan.
Heart Disease (White)	Macmillan.
Diseases of the Chest and the Principles of Physical Diagnosis (Norris and Landis)	Saunders.
Physical Diagnosis (Cabot)	William Wood and Co.
Heart Failure (Fishberg)	Lea and Febiger.

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SECTION V

MINOR ARRHYTHMIAS

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65. Mechanism of heart beat.—It will be remembered that the heart is normally excited to contraction by impulses arising from the sino-auricular node. These impulses pass over the auricular musculature to the auriculoventricular node where they are picked up and transmitted down the bundle of His and its branches to the ramifications of the Purkinje system. As a result of the passage of any single excitation wave over the heart, both auricles contract simultaneously and this contraction of the auricles is followed after a short pause (accounted for by passage of the impulse over the neuromuscular system below the auricles, by the simultaneous contraction of both ventricles. When this impulse formation is regular in rhythm at a rate varying between 60 and 100 per minute and the transmission is undelayed along the channels outlined, the cardiac mechanism is considered to be normal. When that impulse formation is irregular in any manner, is greater or less than 60 to 100 per minute, or the function of impulse formation is taken up by any other structure than the sino-auricular node, the resultant cardiac mechanism is considered to be an arrhythmia.

66. Classification of arrhythmias.—Probably the most acceptable classification of cardiac rhythms and arrhythmias is the following, which is based on the point of origin of pacemaking stimulus:

a. Those arising in the sino-auricular node—

(1) Normal sinus rhythm.
(2) Sinus bradycardia.
(3) Sinus tachycardia.
(4) Sinus arrhythmia.

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(5) Ventricular escape.
(6) Sinus premature contractions.

b. Those arising from within the auricular muscle (ectopic auricular rhythms)—

(1) Premature auricular contractions (auricular extrasystoles).
(2) Auricular tachycardia (paroxysmal).
(3) Auricular flutter.
(4) Auricular fibrillation.

c. Those arising from the auriculoventricular node or bundle of His—

(1) Nodal premature beats (nodal extrasystoles).
(2) Nodal rhythm.
(3) Nodal tachycardia (paroxysmal).

d. Those arising from within the ventricular muscle—

(1) Premature ventricular contractions (ventricular extrasystoles).
(2) Ventricular tachycardia.
(3) Fibrillation of the ventricles.

Of this group of cardiac mechanisms, sinus tachycardia, sinus bradycardia, sinus arrhythmia, the premature contractions and paroxysmal tachycardia are of especial interest to aviation examiners. From a clinico-pathological point of view, the serious auricular arrhythmias and the ventricular mechanisms are of the most importance. However, they are usually the result of cardiac disease rather than the cause, and will rarely be seen in cadet applicants. Therefore, in order to carry out the plan of this manual to a logical conclusion, only the minor arrhythmias which will be commonly seen in this type of work will be discussed here. The more serious auricular and ventricular rhythms are discussed in some detail as clinical entities later.

67. Normal sinus rhythm.—In normal sinus rhythm, the impulse arises in the sino-auricular node (pacemaker) and causes an orderly contraction of auricles and ventricles. The average normal heart rate in healthy adults is about 70 beats per minute for men and about 80 beats per minute for women. In a given individual the pulse rate varies considerably. It becomes more rapid when he passes from the reclining to standing position. It accelerates with excitement and exercise. There is a rough parallelism between the metabolic rate and the pulse rate. The various factors which act on the vagus and accelerator nerves influence the heart rate by acting through these nerves on the sino-auricular node. Further, the pulse rate varies from individual to individual, each person having a basal rate which for him is normal. When

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these rates are not less than 60 and not greater than 100, the pulse rate is considered to be within normal limits.

68. Sinus bradycardia.—Sinus bradycardia differs from normal sinus rhythm only in rate. The impulse arises in the sino-auricular node and passes normally over the conduction channels, but is distinguished by the fact that the rate of impulse formation, and consequently the heart rate, is less than 60 per minute. Sinus bradycardia is caused by anything which disturbs the accelerator-inhibitor balance to produce a vagal predominance. The vagal activity thus produced acts upon the sino-auricular node to slow the heart rate. The principal causes of vagal stimulation are increased intracranial pressure (as seen in meningitis, etc.), drugs, and toxic substances. Digitalis slows the heart rate somewhat but is nowhere near as effective in slowing normal rates as it is in slowing the abnormally fast ones. Bile salts or pigments seen in jaundice also act as toxins and slow the rate. Lowered metabolic states, especially those seen in myxoedema, create a bradycardia. Finally, there is that group of people, in whom no cause can be discovered, who seem to have a normally slow heart rate.

69. Sinus tachycardia.—*a.* Sinus tachycardia, like sinus bradycardia, differs from normal sinus rhythm only in rate. Here, also, the impulse arises in the sino-auricular node and passes normally over the conduction channels, being distinguished only by a rate greater than 100 per minute. It is due to any condition which disturbs the accelerator-inhibitor balance, either a sympathetic stimulation or a removal of vagal inhibition.

b. Normally, the rate of impulse formation at the sino-auricular node is always under a slight continuous vagus inhibition. If this is removed, as can be done by the administration of atropine, the sinus rate becomes more rapid. The pulse rate increases during exercise and excitement, partly due to diminution of vagal tone and partly to increase of accelerator tone. Increased temperature and increased rate of metabolism also cause an increase in pulse rate. Among other causes of simple tachycardia are anemia, hypotension, some types of myocardial disease, infections with fever, exophthalmic goiter, and the effort syndrome.

c. A point which should be noted in passing is that in simple tachycardia the heart rate is influenced by posture, rest, and exercise, whereas in paroxysmal tachycardia, it is not influenced at all by these factors.

d. Paragraph 72*d*, AR 40–105, states, "A heart rate of 100 or over, or of 50 or under, when these are proved to be persistent in the recum-

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bent posture and on observation and reexamination over a sufficient period of time" will be cause for rejection.

70. Sinus arrhythmia.—*a.* There are a number of closely related heart irregularities which, although normal as to point of origin and conduction of the impulses, are due to variations in the rate at which those impulses are generated at the sino-auricular node. These irregularities, grouped under the term "Sinus arrhythmia" are brought about by alterations in vagal tone. The most common type is the irregularity of respiratory origin, in which there is a gradual acceleration of rate during inspiration and a fall of rate during expiration. The immediate cause of this irregularity is vagal stimulation. The act of breathing seems to be sufficient to stimulate the vagus which, acting on the sino-auricular node, causes transient slowing of the whole heart. The condition is recognized clinically by the fact that the heart rate accelerates on inspiration and slows on expiration, this usually disappearing when the breath is held or when the heart's rate is raised by exercise. It can best be recognized by listening to the heart, as in this manner one can detect variations in the rhythm better than with the finger.

b. Other forms also occur. After swallowing, for instance, there may be a vagal slowing of the whole heart. In certain individuals the heart is continually undergoing more or less marked fluctuations in rate which are independent of respiration but due to alterations in vagal tone. Slowing of the heart accompanying fainting spells is of this type also. Finally, sinus arrhythmia is particularly common in nervous individuals, being closely related to other manifestations of nervous instability and irritability, such as exaggerated tendon reflexes and excessive vascular reactions. It is commonly met with in the effort syndrome and sometimes is especially marked in neurocirculatory asthenia. Sinus arrhythmia in itself is of no pathological importance and should not be construed as a "marked cardiac arrhythmia." It is very commonly seen in healthy young adults and is sometimes considered as evidence of good myocardial tonus.

71. Ventricular escape.—This condition cannot be recognized at examination or even clinically (electrocardiographically only), and is without clinical significance. It is the result of slow action of the sino-auricular node. Normally, the regular sinus rhythm is dominant, but if its rate falls below a certain point, or if the automatic rate in the auriculoventricular node rises above the same point, then the ventricles beat spontaneously. These ventricular beats are termed "escaped" ventricular contractions and are truly that, an escape of the ventricle for one beat from the dominance of the normal sinus rhythm.

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The occurrence of ventricular escape is rather uncommon and, insofar as is known at present, of no clinical or prognostic importance.

72. Sinus premature contractions.—This is also of rare occurrence and of no practical interest clinically. It is simply the occurrence of a complete heart beat of sinus origin out of sequence.

73. Auricular premature contractions.—*a.* Auricular premature contractions or extrasystoles may arise from any portion of the auricular muscle. Here, as in the other auricular rhythms, the site of the pacemaker is displaced from the sino-auricular node to a focus somewhere in the auricular muscle. In an auricular premature contraction this new focus emits a single impulse creating a premature auricular contraction, which in turn activates the auriculoventricular node, causing the ventricles to contract in sequence. The ventricles, it will be remembered, when not in a refractory state, respond to each auricular stimulus, normal or abnormal. The auricles having contracted prematurely, the ventricles also participate in the irregularity. The premature auricular beat is followed by a long pause, but the whole period of the disturbance is not equal, as in the case of ventricular premature contractions, to two full cycles of the normal rhythm. Consequently, the fundamental rhythm of the heart is disturbed and from the occurrence of the extrasystole a new rhythmic sequence is set up. It is very difficult to distinguish by examination only between auricular and ventricular premature contractions except by the change in rhythm as manifested by the pause. Here even the most acute observers are often at a loss.

b. Auricular premature contractions are of little clinical significance. Lewis states that "When extrasystoles are found, the heart amongst other organs should be scrutinized closely. If no further signs are detected, the extrasystole can be neglected; if other signs are found, the prognosis will be based on these; the extrasystole has served its purpose and can again suffer neglect." On the other hand, it should be remembered that auricular premature contractions are relatively common in developed mitral stenosis. Also, both experimentally and clinically, auricular premature contractions, auricular tachycardia, auricular flutter, and auricular fibrillation seem to be intimately related and numerous transitions between the above disorders have been observed. Patients who suffer from attacks of paroxysmal auricular tachycardia frequently show auricular premature contractions between attacks. Auricular premature contractions are also common after attacks of paroxysmal auricular fibrillation, and transitions between auricular flutter and fibrillation are not uncommon. Finally, clinically, in a single patient with asso-

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ciated myocardial disease it is not uncommon to see at first occasional auricular premature contractions; later, multiple auricular premature contractions (short paroxysms of auricular tachycardia); still later, paroxysmal auricular fibrillation; and finally, permanent fibrillation with consequent cardiac failure. So from the intimate relationship that exists between these associated disorders it seems likely that they are all produced by similar pathological changes and that, on occasion, auricular premature beats may be of some significance.

c. Subjectively patients may complain that their hearts have "skipped a beat", "turned over", "jumped." Often these symptoms can be very annoying and patients sometimes complain of accompanying pain. Recognition without electrocardiographic aid is made through the occurrence of an extra heart beat, best described as "coupling". The extra beat is easily detected by the stethoscope. Auricular premature contractions are usually abolished by exercise and aggravated by caffeine and nicotine.

74. Auricular paroxysmal tachycardia.—*a.* Paroxysmal tachycardia is an abnormal heart rhythm caused by a rapid and regular succession of auricular premature contractions. It is a condition in which the normal rhythm of the heart is submerged by a series of rapid and regular beats of extra-sinus origin which vary in rate from 100 to 200 per minute. This series starts abruptly, stops abruptly, and does not vary in rate at all while the new focus in the auricular muscle has control. The first beat of the series is premature and the last beat is followed by a pause, following which the sino-auricular node again takes over and the heart again assumes a normal sinus rhythm. In duration, the tachycardia lasts from a few beats to hours or days. Very rarely, it lasts two weeks or longer.

b. While paroxysmal tachycardia may occur at any age, the highest incidence is between the ages of twenty and thirty. In at least one-half the cases, the etiology is indefinite. Some appear to follow the acute infections of childhood. The only infection which appears at all common in the history of these cases is rheumatic fever. A common pathological lesion found in the heart muscle is a localized or diffuse fibrosis. Most cases show no evidence of valvular disease. When valvular disease is present, mitral stenosis is the predominant lesion found. A certain proportion of cases show a moderate degree of cardiac enlargement and a limited response to exercise.

c. In patients subject to attacks of paroxysmal tachycardia, a sudden strain or emotional disturbance often seems to induce an attack. In many other cases, no such inciting cause can be found. The symp-

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toms are variable and in the main depend upon the duration of the attack, the ventricular rate, and the state of the heart muscle. When attacks are brief, they may pass unnoticed, but if they last over one-half hour they are almost sure to produce symptoms. Usually, the patient is made conscious of the onset of the rapid heart action by a sense of discomfort or a fluttering sensation in the chest. If the attack persists, a feeling of exhaustion, coldness, and sweating are often experienced. Then gastric symptoms may set in within an hour or so, with flatulence, salivation, nausea, and vomiting. Anginal attacks may occur, and vary much in severity with precordial pain, hyperalgesia and hyperesthesia, tender muscles, and a sense of constriction around the chest. In some cases where the rate has been very rapid or the musculature weak, the signs and symptoms of congestive cardiac failure appear. The heart dilates and the apex beat may be found to steadily progress beyond the nipple line. There is progressive engorgement of the veins of the neck, cyanosis, and enlargement and tenderness of the liver. The attack may terminate in progressive failure with ascites, anasarca, and death. Paroxysmal tachycardia is one of the few conditions in which there may be an acute dilatation of the heart. In this case, however, the dilatation is the result, not the cause, of the rapid heart rate.

d. The attacks in paroxysmal tachycardia are often terminated by a change in posture, the sipping of cold liquids, vomiting, etc. Pressure on eyeballs, carotid sinus, or on the vagus occasionally is effective. These procedures may be repeatedly effective in certain individuals, while in others they may all fail. In this latter circumstance the drug of choice is acetyl-beta-methylcholine, commonly called mecholyl. The dosage is 20 to 40 milligrams subcutaneously. Results are usually immediate, or follow a second dose which should be given after a 15-minute interval. Digitalis is of no value in the treatment of this condition.

e. When the attack does stop, it ceases as suddenly as it started and almost immediate relief is experienced. The abrupt beginning, abrupt ending, unvarying rate even under conditions of rest, exercise, or emotion, and the rate between 100 and 200 make the differentiation from sinus tachycardia. Simple tachycardia is of gradual onset, gradual cessation, and varies widely with rest, position, exercise, and emotion, and rarely exceeds a rate of 150.

f. The immediate prognosis for the attack is good. Although deaths during paroxysms have occurred, the vast majority endure the attacks surprisingly well. The ultimate prognosis is based on an estimate of the condition of the cardiac muscle as in other heart

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conditions, that is to say, upon the other evidences of myocardial disease. The attacks themselves usually indicate some degree of myocardial disease, and in view of this fact, paragraph 31*a*, AR 40-110, states that a history of paroxysmal tachycardia disqualifies both on the original and subsequent examinations.

75. Nodal rhythms.—*a.* In general, the nodal rhythms have the same signs and symtoms as do the auricular rhythms and clinically cannot be distinguished from them. The differentiation is made entirely by the electrocardiogram. Physiologically, the function of the pacemaker is taken over by the auriculoventricular node or the first portion of the bundle of His above the bifurcation for a variable period of time. With each impulse arising from these junctional structures, both the auricles and ventricles contract simultaneously. The clinical results of this simultaneous contraction are not distinguishable from the auricular rhythms without mechanical aids except in the one condition, auricular fibrillation. It should be noted in passing that nodal tissue does not give rise to fibrillation.

b. Nodal premature contractions have essentially the same causes, significance, and physical signs as do those of the auricular type. In fact, the relationship is so close that many authorities prefer to group both auricular and nodal premature contractions into one classification, and call them supraventricular premature contractions.

c. Nodal rhythm is that condition in which the dominant rhythm of the heart originates in the auriculoventricular node rather than in the sino-auricular node. The rate here is somewhat slower than that of normal sinus rhythm, but otherwise is distinguishable from it only by means of the electrocardiograph. Occasionally, the nodal tissue will emit impulses at such a rate as to constitute a tachycardia. This condition is known as nodal tachycardia, and differs only from nodal rhythm in rate. Here again, the diagnosis is one for electrocardiography, it being impossible to detect clinically. These rhythms are not uncommonly found following toxic doses of digitalis or atropine.

76. Ventricular premature contractions.—*a.* Ventricular premature contractions arise from some ectopic focus in the ventricles and disturb the normal rhythm by occurring before the rhythmic stimulus from the auricles is due. The disturbance of rhythm is, as a rule, limited to the ventricles, the auricles continuing to contract at their normal rhythm. The impulse from the succeeding normal auricular contraction usually reaches the ventricles during the refractory period that accompanies their premature beats, and consequently causes no ventricular contraction. Ventricular premature

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contractions are, therefore, followed by a pause which lasts until the next stimulus arrives from the auricles. Since the auricular rhythm is not disturbed, the time intervening between the normal ventricular systole immediately preceding and immediately following the premature contraction is equal to the interval between two normal beats. There is, therefore, a full compensatory pause after the premature contraction, thus differing from the auricular premature contraction in which the compensatory pause is shortened.

b. Clinically, ventricular premature contractions are of little importance. In the largest group, no evidence of heart disease can be found. In another important, but smaller group, they are relatively common in patients suffering from myocardial degeneration. Thus, they are not uncommonly associated with developed mitral stenosis, free aortic regurgitation, coronary artery disease, and chronic hypertension. In another or toxic group, premature contractions are commonly found. In some cases, they seem to be associated with excessive use of tobacco, or of coffee, dietary excesses, and toxic doses of digitalis. They occasionally occur in acute infectious diseases, later disappearing.

c. In considering prognosis, ventricular premature beats are of little significance. As in the case of auricular premature contractions, ventricular premature contractions are to be ignored when they occur in the absence of other signs of heart disease. When they occur in conjunction with other signs of heart disease, the prognosis is to be based on those signs, and the ventricular premature beats are to be disregarded.

77. Recommended texts:

The Mechanism and Graphic Registration of the Heart Beat (Lewis)	Shaw and Sons.
Diseases of the Heart (Lewis)	Macmillan.
Heart Disease (White)	Macmillan.
Clinical Disorders of the Heart Beat (Lewis)	Shaw and Sons.

Section VI

ETIOLOGICAL CONSIDERATIONS

	Paragraph
Importance	78
Incidence	79
Factors	80
Recommended texts	81

78. Importance.—*a.* The importance of etiology in the consideration of heart disease is best illustrated by the attitude of the American

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Heart Association in establishing standards of nomenclature. The association has divided the complete diagnosis of cardiac disturbances into four elements: etiological, anatomical, physiological, and functional capacity.

b. In a large measure, knowledge of the etiology will, in addition to completing the diagnosis, control both the type of treatment given and the prognosis. An understanding of etiology will also aid in the prophylaxis and prevention of heart disease. Without this knowledge these measures would be merely blind thrusts.

79. Incidence.—*a.* White estimates that 2 percent of the population have a sufficient amount of cardiac disease to produce signs and symptoms. Another authority maintains that 90 percent of all heart disease is caused by the four major conditions: rheumatic fever, syphilis, hypertension, and arteriosclerosis. The remaining 10 percent of heart diseases are caused by such miscellaneous conditions as congenital malformations, thyroid disease, acute and subacute bacterial endocarditis, diphtheria, toxic heart disease, and the damage done by pulmonary hypertension, anemias, trauma, and neoplasms. A large factor in this 10 percent will have to be stated as simply unknown.

b. The facts herein mentioned are of the utmost importance to the student of cardiology. The mere knowledge of the percentages mentioned simplifies the approach to diagnosis greatly. Given a case presenting the signs and symptoms of a cardiac condition, the chances are immediately in your favor, nine to one, that it will fall into one of the four major types mentioned. Combine this with the distribution of etiological causes by age groups (par. 80*a*) and a case can be approached with the etiological portion of the diagnosis practically made. Two examples will suffice. A young man of twenty has all the signs and symptoms of heart trouble. Immediately you know that the chances are overwhelmingly in favor of rheumatic fever as the causative factor. Given a man sixty with similar signs and symptoms you know the chances are overwhelmingly in favor of either arteriosclerosis or hypertension as the cause. Simple examining procedures will make the differentiation. Of course, the other 10 percent of causes must be kept in mind in following such a procedure.

c. In the past three decades, the recorded incidence of deaths due to heart disease has increased by leaps and bounds. It now ranks ahead of all of the diseases as the cause of death, including such serious factors as tuberculosis, pneumonia, and the malignancies. The Surgeon General's report for the Army, 1936, listed disease of the coronary

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arteries alone as fourth ranking cause of death, exceeded only by automobiles, suicides, and airplanes. The Public Health Reports for 1936 give the death rate from cardiac causes in 1932 as 221.9 per 100,000. In 1936 it was 276.7 per 100,000, an increase of 24.7 percent in 4 years. The nearest other rates in 1936 were cancer, given as 116.4 per 100,000 and pneumonia as 89.1.

d. This rise in rate is popularly explained by the increase in longevity that has occurred in the past 30 years. A closer analysis reveals that this increase in longevity is really a decrease in the fatal diseases of childhood, plus a parallel decrease in the incidence of the infectious diseases. Both of these are due to the improved preventive health measures now in effect. In other words, people are living longer, so that now they die of the old age diseases instead of the childhood and middle age diseases. Another and very potent factor to be considered is the improved method of reporting heart disease. Physicians are now better educated and more heart conscious, and many cardiac conditions previously reported as something else are now recognized as heart disease.

e. In an analysis of 450 deaths from heart disease in the District of Columbia for 1934, the following figures, very well illustrate the percentages mentioned earlier in this section:

Arteriosclerosis (hypertensive)	276	Congenital	9
Rheumatic fever	60	Thyroid	6
Syphilis	54	Others	3
Bacterial	21	Undetermined	21

80. Factors.—There are several more or less related factors in the consideration of etiology that should be mentioned at this time. They are in probable order of importance: age, climate, race, heredity, sex, and social status.

a. Age.—The various heart diseases fall readily into age groups. Rheumatic fever is definitely a disease of youth, the cardiac end results being seen between the ages of five and forty-five. The peak of incidence for the rheumatic cardiac diseases occurs in the third decade. Cardiac syphilis due to the period of latency of from ten to twenty years which follows the primary lesion, is a disease of late middle life, thirty-five to fifty. The peak of incidence here is in the fifth decade. Hypertension and arteriosclerosis are diseases of later life. The first appearance of these degenerative diseases, with rare exceptions, is seen in the fifth decade, incidence continuing into the advanced ages. The peak of occurrence is seen in late sixth and early seventh decades.

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Of the remaining 10 percent of causative factors, all but the congenital diseases are those with a rather wide age spread. However, their cardiac results are manifested chiefly in early adulthood and middle life. The importance of these age groupings has been mentioned in connection with their use in arriving at cardiac diagnoses.

b. Climate.—(1) Climate is of importance under both geographical distribution and seasonal variation. In the north, and especially in the northeastern United States, rheumatic fever is of major importance. In the south it is of rather rare occurrence. In the south, syphilis ranks high in importance as a cause of cardiac pathology, whereas in the north and northeast it is of rather minor importance. The degenerative diseases are of rather universal distribution, having little geographical variation. It should be noted in this connection that the degenerative diseases seem to be somewhat less in the Tropics. Geographical distribution is well illustrated by the following figures, taken from the Public Health Reports above mentioned. Cardiac death rates for 1936 for the areas reported were 276.7 per 100,000 population. All of the individual States having death rates of 250 per 100,000 or more were northern States, with the sole exceptions of Maryland and the District of Columbia. Those States having rates of less than 200 per 100,000 were predominantly southern. Rheumatic fever is the probable cause.

(2) Seasonal variation is well illustrated in the same reports. The period January to March showed the greatest number of deaths. October to December was second in incidence, April to June third, and July to September showed the least. Apparently, winter and the rigors of cold weather have a very definitely deleterious effect on the incidence of cardiac diseases.

c. Race.—The importance of race is shown by the fact that negroes have well over two times as much heart disease per unit of population as do whites. Here, syphilis is the chief offender while hypertension runs a close second. Rheumatic fever is relatively rare in the colored population. The large incidence of syphilis in negroes somewhat explains the fact that the bulk of luetic heart disease is found in the south.

d. Sex.—Sex is of little importance, the general incidence being practically the same between the two sexes.

e. Heredity.—Heredity in heart disease is a much disputed point. Considerable difference of opinion exists but it is safe to say, as in tuberculosis, that the tendency toward the various degenerative diseases is believed to be inherited.

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f. Social status.—Social and financial status has a common effect on such diseases as rheumatic fever and syphilis. Rheumatic fever is definitely increased in, but not entirely limited to, those families suffering the lowered hygiene, overcrowding, and poor nutrition incident to the lower classes. Likewise, syphilis is found with a somewhat greater frequency in the classes less fortunate financially.

81. Recommended texts.

Heart Disease (White)---------- Saunders.
Current literature, especially periodic reports of the United States Public Health Service.

Section VII

NOMENCLATURE FOR CARDIAC DIAGNOSIS*

	Paragraph
Classification of diagnostic titles	82
Recommended text	83

82. Classification of diagnostic titles.—*a.* From a study of the following tables of diagnostic titles it will be seen that a complete diagnosis is intended to include not only a statement of the various structural changes which may be found in the heart or aorta, but also one concerning the cause of such changes, one indicating any disturbance of physiological function which may have resulted, and finally, one to indicate the functional capacity of the patient with heart disease.

b. Such a comprehensive diagnosis must require careful consideration of every aspect of the case and will afford a sound basis for prognosis and effective treatment.

c. Each diagnosis should include one or more titles from each of the groups, (1), (2), (3), and (4). Should a diagnosis of normal heart be made and a condition which might cause heart disease be found, the patient is placed in group (6) with a statement of the possible etiological factor. Should the diagnosis be in doubt, the patient is retained for further consideration under group (5).

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*This entire section is reproduced verbatim from the reference handbook "Criteria for the Classification and Diagnosis of Heart Disease," published by the Heart Committee of the New York Tuberculosis and Health Association, Inc., 386 Fourth Avenue, New York, New York. Dr. Ernest P. Boas, Chairman of the New York Heart Association, has kindly granted permission for the inclusion of this section in this manual. Dr. Harold E. B. Pardee, Chairman of the Criteria Committee, has very kindly furnished an advance copy of the revised nomenclature as it will appear in the new fourth edition of the handbook.

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(1) *Etiological diagnosis.*
(*a*) Anemia.
(*b*) Arteriosclerosis.
(*c*) Bacterial infection (specify if possible).
(*d*) Congenital anomaly.
(*e*) Hypertension.
(*f*) Hyperthyroidism.
(*g*) Hypothyroidism.
(*h*) Neoplasm.
(*i*) Other etiological factor (to be specified).
(*j*) Psychoneurosis.
(*k*) Pulmonary disease (to be specified).
(*l*) Reflex action.
(*m*) Rheumatic fever.
(*n*) Syphilis.
(*o*) Thoracic deformity.
(*p*) Toxic agent (specify if possible).
(*q*) Trauma.
(*r*) Unknown.
(2) *Anatomical diagnosis.*
(*a*) *Diseases of aorta and pulmonary arteries.*
1. Aneurysm (specify location).
2. Aortitis.
(a) Without dilatation.
(b) With dilatation.
3. Arteriosclerosis of aorta.
(a) Without dilatation.
(b) With dilatation.
4. Arteriosclerosis of pulmonary arteries.
5. Congenital anomaly (specify if possible).
6. Embolism of pulmonary arteries.
7. Injury of (specify location).
8 Other disease of aorta (specify lesion).
9. Other disease of pumonary arteries (specify lesion).
10. Rupture (spontaneous).
11. Thrombosis of aorta.
12. Thrombosis of pulmonary arteries.
(*b*) *Conorary arteries.*
1. Arteriosclerosis of coronary arteries.
(a) With narrowing.
(b) With occlusion.
2. Arteritis of coronary arteries.
3. Congenital anomaly of coronary arteries.

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4. Embolism of coronary artery.
5. Injury of coronary artery (specify character of lesion).
6. Other disease of coronary arteries (specify).
7. Periarteritis nodosa of coronary arteries.
8. Stenosis of coronary ostium.
9. Thrombosis of coronary artery.

(*c*) *Diseases of myocardium* (including conduction system and heart as a whole).

1. Aneurysm of heart (specify location).
2. Atrophy of heart.
3. Congenital anomaly (specify lesion if possible).
4. Degeneration of myocardium (specify type, if possible).
5. Enlargement of heart (chambers involved may be specified).
   (a) Dilatation.
   (b) Hypertrophy.
6. Fatty infiltration of heart.
7. Fibrosis of myocardium.
8. Infarct of myocardium.
   (a) Recent.
   (b) Healed.
9. Injury of heart (specify character of lesion).
10. Myocarditis, active.
11. Myocarditis, inactive.
12. Neoplasm of heart (specify type).
13. No structural disease.
14. Other structural disease of heart (specify lesion).
15. Rupture of myocardium (specify location).
16. Thrombosis within heart (specify chamber affected).
17. Undiagnosed structural disease (specify location, if possible).

(*d*) *Diseases of endocardium and valves.*

1. Congenital anomaly (specify lesion, if possible).
2. Endocarditis, acute bacterial (specify organism).
3. Endocarditis, indeterminate.
4. Endocarditis, subacute bacterial (endocarditis lenta) (specify organism).
5. Injury of endocardium or valve (specify lesion).
6. Mural endocarditis.
7. Mural thrombosis.
8. Other structural disease (specify location, if possible).
9. Rupture of valve (specify valve).

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10. Sclerosis of valve (specify valve lesion).
11. Valvulitis, active (specify deformity, if any).
12. Valvulitis, inactive (specify deformity, if any).
13. Valvular deformity.
(a) Aortic insufficiency.
(b) Aortic stenosis.
(c) Mitral insufficiency.
(d) Mitral stenosis.
(e) Pulmonic insufficiency.
(f) Pulmonic stenosis.
(g) Tricuspid insufficiency.
(h) Tricuspid stenosis.

(*e*) *Diseases of pericardium.*
1. Classification of pericardium.
2 Congenital anomaly of pericardium (specify lesion).
3 Hemopericardium.
4. Hydropericardium.
5. Injury of pericardium (specify character of lesion).
6. Neoplasm of pericardium.
7. Pericarditis, acute.
(a) Fibrinous.
(b) Serofibrinous.
(c) Suppurative.
8. Pericarditis, chronic.
(a) Adhesive without constriction.
(b) Constrictive.
(c) Serous.
9. Pneumopericardium.

(3) *Physiological diagnosis.*
(*a*) *Cardiac mechanism.*
1. Arrhythmia (undiagnosed).
2. Auricular fibrillation.
(a) Paroxysmal.
(b) Persistent.
3. Auricular flutter.
(a) Paroxysmal.
(b) Persistent.
4. Auriculoventricular block.
(a) Prolonged conduction time.
(b) Incomplete.
(c) Complete.

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5. Auriculoventricular nodal rhythm (junctional rhythm).
6. Bundle branch block.
7. Other arrhythmia (specify).
8. Paroxysmal tachycardia.
(a) Auricular.
(b) Auriculoventricular nodal (junctional).
(c) Ventricular.
(d) Unknown origin.
9. Premature contractions.
(a) Auricular.
(b) Auriculoventricular nodal (junctional).
(c) Ventricular.
(d) Unknown origin.
10. Sinus arrest.
11. Sinus arrhythmia.
12. Sinus bradycardia.
13. Sinus rhythm.
14. Sinus tachycardia.
15. Ventricular escape.
16. Ventricular fibrillation.
17. Ventricular flutter.
18. Wandering pacemaker.
19. Valvular incompetence.
(a) Aortic incompetence.
(b) Mitral incompetence.
(c) Pulmonic incompetence.
(d) Tricuspid incompetence.

(*b*) *Clinical syndromes.*
1. Adams-Stokes syndrome.
2. Anginal syndrome.
3. Cardiac insufficiency.
4. Carotid sinus syndrome.
5. Pulsus alternans.
6. Paroxysmal dyspnoea.
7. Paroxysmal pulmonary edema.

(4) *Functional capacity.*

(*a*) *Class I.*—Patients with a cardiac disorder without limitation of physical activity. Ordinary physical activity causes no discomfort.

(*b*) *Class II.*—(Formerly II-A*.) Patients with a cardiac disorder with slight to moderate limitation of physical activity. Ordinary physical activity causes discomfort.

*The correspondence with the former classes II-A, II-B, and III is only in the case of patients with organic heart disease.

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(*c*) *Class III.*—(Formerly II-B*.) Patients with a cardiac disorder with moderate to great limitation of physical activity. Less than ordinary physical activity causes discomfort.

(*d*) *Class IV.*—(Formerly III*.) Patients with a cardiac disorder unable to carry on any physical activity without discomfort.

(5) *Possible heart disease.*—Patients who show abnormal signs or symptoms referable to the heart but in whom the diagnosis of heart disease is uncertain should be diagnosed as "Possible heart disease, class E."

(6) *Potential heart disease.*—Patients without heart disease, whom it is advisable to follow because of the presence or history of an etiological factor which might cause heart disease, should be diagnosed as "Potential heart disease, class F." In such cases the etiological factor should be stated.

(7) *Therapeutic classification.*

(*a*) *Class A.*—Patients with a cardiac disorder whose ordinary physical activity needs no restriction.

(*b*) *Class B.*—Patients with a cardiac disorder whose ordinary physical activity needs no restriction but who should be advised against unusually severe or competitive efforts.

(*c*) *Class C.*—Patients with a cardiac disorder whose ordinary physical activity should be moderately restricted, and whose more strenuous habitual efforts should be discontinued.

(*d*) *Class D.*—Patients with a cardiac disorder whose ordinary physical activity should be markedly restricted.

(*e*) *Class E.*—Patients with a cardiac disorder who should be at complete rest or confined to bed.

d. This classification of heart disease for diagnostic nomenclature is probably the most scientific yet presented to the practice of cardiology and the most applicable. This system of diagnostic nomenclature is recommended to all flight surgeons. However, it is to be noted that this nomenclature is not official in the United States Army and at the present time in no way replaces section VII, AR 40–1025. It is included for the information of and recommended for special consideration by the Reserve officers of the correspondence course.

83. Recommended text.

Criteria for the Classification and Diagnosis of Heart Disease, 4th Edition—New York Tuberculosis and Health Association.

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*The correspondence with the former classes II-A, II-B, and III is only in the case of patients with organic heart disease.

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CHAPTER 3

DISEASES SEEN IN APPLICANTS FOR FLYING TRAINING

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SECTION I

CONGENITAL HEART DISEASE

	Paragraph
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84. Definition and classification.—Congenital heart disease is that complex group of cardiac conditions caused by anomalous development of the various structures of the heart and great vessels during the intra-uterine period. They are rather rare and seen mostly at birth or in the first few years of life. Occasionally an individual lesion is so slight that the afflicted person will lead a fairly normal life, but for the most part the victims of these whims of nature do not reach adulthood. Numerous lesions and combinations of lesions exist. The more common of these only will be mentioned. In the order of frequency of occurrence they are: patent foramen ovale, defective ventricular septum, patent ductus arteriosus, defects of the pulmonary orifice, valvular anomalies, coarctation of the aorta, and transposition of the large vessels.

85. Patent foramen ovale.—This condition, in which the opening between the right and left auricles fails to close at birth, is one of

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the most common of congenital cardiac abnormalities. This defect is usually associated with congenital stenosis of the pulmonary artery. If not associated with other abnormalities, it usually does not give rise to noticeable symptoms and may be overlooked entirely.

86. Defective ventricular septum.—This is a very common form of congenital malformation. Usually only part of the septum is involved, but it may be complete. It is found most commonly in the upper portion of the interventricular septum and usually is associated with other cardiac abnormalities. Cyanosis is a prominent feature in this condition, due to admixture of blood. Most cases fail to reach adulthood.

87. Patent ductus arteriosus.—This duct should close during the first 2 weeks of extra-uterine life, although it is not unusual to find it remaining patent. In its patency it is associated usually with other abnormalities, especially coarctation of the aorta. Alone, it is of little consequence—very little venous blood reaching the systemic circulation. Patients have a good, although not normal, expectancy.

88. Defects of pulmonary orifice.—Stenosis is the most common of the defects of this orifice. Atresia, or absence of the pulmonary orifice, is much less frequently encountered, but is a much more serious condition. Usually, both are associated with other anomalies and the prognosis in general is not good.

89. Valvular anomalies.—Valvular anomalies usually consist of such entities as bicuspid or quadricuspid valves and, unless complicated by endocarditis, are of no importance. They are rarely detected prior to autopsy. Any one or all of the valves of the heart may show congenital defects. The right side is more frequently involved than the left. Some authorities consider valvular defects entirely the result of foetal endocarditis.

90. Coarctation of aorta.—This is a serious and disabling condition, frequently missed, in which there is a localized narrowing of the aorta in the vicinity of the insertion of the ductus arteriosus. In the pure infantile type the maximum age reached is less than ten months, while in the adult type the average age is nearer thirty. Very few of these cases will be in good enough physical condition to allow presentation of themselves for enlistment. All show a disqualifying degree of hypertension.

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91. Transposition of large vessels of heart.—This is not a rare condition, but is one in which the diagnosis is very difficult. Uncomplicated by other associated defects, there are no signs or symptoms which will give a clear diagnosis.

92. General considerations.—*a.* In general, the serious congenital heart diseases present the common symptoms of dyspnoea and disability. Cyanosis, clubbing, and malnutrition are the common signs. These are complicated by the various arrythmias, blocks, and degrees of congestive failure which accompany the more advanced stages. Exact diagnosis is unusually difficult prior to post mortem, and even the best of cardiologists are frequently at a loss to make a diagnosis more specific than simply congenital heart disease. This is true because of the varying degrees of defects and the multiplicity of possible combinations. It is to be emphasized again that the defects most frequently exist in the heart in groups and that such cardiac defects are usually acompanied by other and more evident extra cardiac anomalies.

b. The most important of these groupings mentioned is the Tetrology of Fallot, which is the most common finding in adults with the cyanotic type of congenital heart disease. The component parts of the tetrology are pulmonary stenosis, interventricular septal defect, dextro-position of the aorta, and hypertrophy of the right ventricle. For a complete discussion of the various other combinations possible, the student is referred to the recommended texts.

c. In examination for aviation, congenital cardiac diseases have a very small part. They are comparatively rare and, when serious enough to be of importance, they will be of such a disabling nature that the applicant could not otherwise be accepted. When of a minor nature, they will most frequently be missed by even the most acute observers. Even though the minor types present a good prognosis, when they can be diagnosed the individual must be rejected for flying training on the basis of organic heart disease.

93. Recommended texts.

Atlas of Congenital Cardiac Disease (Abbott)	American Heart Association, 1936.
Heart Disease (White)	Macmillan.
Clinical Heart Disease (Levine)	Saunders.
Diseases of the Heart (Lewis)	Macmillan.

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SECTION II

RHEUMATIC FEVER

	Paragraph
General	94
Etiology	95
Pathology	96
Signs and symptoms	97
Prognosis	98
Treatment	99
Recommended texts	100

94. General.—*a.* Paragraph 31*a* (7), AR 40–110, states that "a history of recurrent attacks of any of the rheumatic group" should be considered as disqualifying for flying. The rheumatic group indicated therein includes all of the entities which form that rather complex group of conditions having a common pathological background of rheumatic inflammation. The principal members of this group are the acute and subacute manifestations of polyarthritis, chorea, and pancarditis, plus the almost inevitable end result, chronic rheumatic valvular disease. To these should be added the less common entities which are discussed below as atypical manifestations.

b. Rheumatic fever is better understood if the condition is thought of as "the rheumatic state"; a polysystemic invasion of the body by the causative rheumatic agent, comparable to that of tuberculosis and syphilis. A fairly close parallel can be drawn between these three diseases in the matter of their various systemic manifestations to a single type of inflammation. All have a principal primary location; rheumatic fever in the joints, tuberculosis in the lung tissue, syphilis in the genito-urinary tract. All three likewise affect certain other systems as each advances, and all three are capable of manifesting themselves on occasion at points other than the usual primary location.

c. While rheumatic fever attacks principally the joints, heart, and central nervous system, practically all of the other systems of the body have been reported at one time or another as being involved. It is believed that many of the vague illnesses of children are merely manifestations of low-grade rheumatic inflammation of these other systems. The growing pains (myositis), unexplained vomiting (gastrointestinal tract), unexplained nosebleeds (vascular system), erythema multiforme and rheumatic nodules (skin) would seem to more or less confirm this opinion. Various writers have pointed out the apparent relationship between these conditions and "the rheumatic state." However, it should be said in all fairness that the

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decision as to whether these borderline conditions are rheumatic or not is chiefly one of opinion, and still open to considerable controversy.

95. Etiology.—Rheumatic fever is definitely a disease of childhood and youth. White states that "it is one of the chief scourges of youth, crippling and killing children and young adults." It is but rarely seen before the age of five, and its onset rarely noted after the age of twenty. The causative agent is unknown, and not only is the organism unknown, but the method of transmission and the portal of entry into the body are as yet undetermined. The best contemporary opinion holds that the organism is either streptococcus or a virus, and that the portal of entry is through the nose and throat. The disease is found chiefly in the temperate zones, New England and the British Isles being definite foci. Not only do cold, damp climates seem to favor the disease, but the cold, damp months, late winter and spring, show the greatest peak of incidence in this country. The disease is no respector of persons, although the poorly nourished, overcrowded, underprivileged classes, with definitely poorer general resistance and hygiene, seem more susceptible. There is no definite evidence that the disease is either contagious or infectious.

96. Pathology.—*a.* The pathology of rheumatic fever is that of an acute inflammatory reaction to an invading organism or toxin. The typical rheumatic lesion is a submiliary nodule situated close to the blood vessels, in interstitial connective tissue. This lesion, or modifications of it, are found in all rheumatic pathology. In the joints there is periarticular involvement, synovial involvement, and synovial fluid changes. The periarticular tissues are swollen and edematous. The synovial surfaces are acutely inflamed, edematous, and present the typical nodules in great numbers, plus the proliferative cell response to inflammation. The synovial fluid is increased in amount, cloudy, and contains many exudative cells. The entire process in the joints, insofar as is known, is bacteria free. In chorea there is a widespread distribution of the perivascular lesion throughout the brain, plus an increase in neuroglia tissue.

b. It should be mentioned in passing that the allergy theory of inflammatory reaction in the joint surfaces very readily explains the presence of inflammation without organisms. Frequently, polyarthritic responses are noted in the presence of organisms invading tonsils, sinuses, and neighboring structures. It is believed that herein lies a large part of the solution of the problem of cause and effect in rheumatic fever. It may be possible, however, that circulating tox-

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ins, or a type of organism which has as yet escaped detection, is the causative factor.

97. Signs and symptoms.—*a.* In discussing signs, symptoms, and diagnosis of acute rheumatic fever, both typical and atypical cases must be considered. Pancarditis and chronic rheumatic valvular disease are discussed in paragraphs 103 and 118 to 120.

b. The typical cases are either those of acute migrating polyarthritis or chorea. Considerable doubt exists upon the idea that pure uncomplicated chorea is an important cause of chronic rheumatic valvular disease. However, for the present time, it is believed best to accept the concensus of cardiologists that chorea is a definite part of the rheumatic state, leading to valvular involvement.

c. Rheumatic polyarthritis is characterized by a shifting involvement of the joints, aptly termed fugitive polyarthritis, accompanied by fever, sweating, and leucocytosis. The large joints are most often involved, and the inflammation is characterized by exquisite pain, tenderness, swelling, heat, and redness. The involvement of any one joint is of short duration, lasting from 3 days to a week, and then shifting to some other large joint. Usually, by the time one joint is subsiding, another is showing early involvement. The smaller joints are involved rather infrequently. The severity of the fever follows the severity of the joint involvement in general, as does the sweating and the leukocytosis. The other signs and symptoms of general malaise are also an accompaniment. The pulse is usually rapid and is out of proportion to the temperature, usually being much faster than the degree of temperature would indicate, and the tachycardia often lasts a considerable time after the fever and other manifestations have subsided.

d. Chorea is characterized by typical purposeless muscular movements of all degrees of severity. It is rarely accompanied by fever of any degree, and the pulse rate is very little accelerated. Fairly frequently, chorea and polyarthritis occur together.

e. The atypical cases of rheumatic fever are chiefly those subacute involvements which are not severe enough to produce the typical picture. Many are overlooked because the possibility of rheumatic fever in an atypical form is not considered. All grades of fever, leukocytosis, and tachycardia can be produced without a characteristic accompanying polyarthritis. In any case presenting symptoms and signs of an indeterminate low grade inflammatory reaction with malaise, loss of appetite, listlessness, fatigue, sweating, and an apparent anemia, rheumatic fever should always be considered until the cause is proved something else. Likewise, any

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child having indeterminate inflammatory signs, who has a persistant tachycardia, should always be under suspicion. Even listlessness, poor appetite, failure to make proper weight gains, and, in fact, all illnesses of more than temporary nature in children should be considered as possibly rheumatic fever, until proved definitely some other entity. This is especially true in regions where rheumatic fever is more or less endemic.

f. There are several other signs and symptoms which are helpful in making a diagnosis of rheumatic state. Frequent nontraumatic epistaxis is a manifestation, as is also frequent vomiting which lacks both apparent cause and nausea. Indefinite abdominal pain and tenderness are considered as other manifestations. Some more commonly known signs are growing pains, rheumatic nodules, erythema multiforme, and precordial ache. Of these, the rheumatic nodules alone are definitely diagnostic of the disease, the others being merely suggestive. Indeed, rheumatic nodules occur only in the presence of severe involvement.

98. Prognosis.—The prognosis of the rheumatic state varies with the number of previous attacks and the severity of the involvement. One characteristic of the disease is a tendency to recur and a majority of cases suffer more than one exacerbation. Some authorities believe that once the rheumatic process becomes manifest, the system is never again free of it, and that each recurrence is merely an exacerbation of the dormant, smoldering condition. Each attack leaves its share of damage in the heart, the amount varying with the severity of the process. Therefore, the more severe and the more frequent the recurrences, the more the heart is involved. Each new attack adds insult to previous injury and with each new attack the prognosis is that much less favorable. Rarely does a patient die in the acute involvement, although it does infrequently happen that a fulminating case may terminate fatally in the first attack. Usually death occurs as the result of the chronic rheumatic valvular disease, which is the ultimate end of the heart involvement. It is fairly safe to assume that every attack of rheumatic fever involves the heart in some degree, although that degree may not be severe enough to produce signs or symptoms.

99. Treatment.—The treatment of a typical acute attack involves both rest and symptomatic relief. Rest is of the greatest importance and is the only constructive thing that can be done to minimize heart damage. Prolonged bed rest for a period of at least 30 days following complete subsidence of fever and the return of pulse to normal is the rule. This is followed by a period of convalescence featured by a

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gradual return to activity. Salicylates given in dosage of 0.1 gram of the sodium salt per kilogram of body weight per day in divided doses will give relief. In all but the most serious cases, relief will be fairly complete by the end of 48 hours' medication. Following this, maintenance dosage of not less than 0.3 gram t. i. d. is required for an extended period. Chorea, likewise, is best treated by rest and symptomatic relief. Here again rest should be absolute and follow the same general rules laid down above. Sedatives give symptomatic relief to some degree, luminal and tincture of stramonium being those recommended. Salicylates have no appreciable effect in chorea. Other signs and symptoms usually warrant only symptomatic relief, although when rheumatic fever is suspected, rest should be instituted and carried out assiduously until pulse and fever criteria have been met, or some other diagnosis definitely made. In all cases, foci of infection should be removed after the subsidence of tachycardia and fever, but never during the acute states.

100. Recommended texts.

Clinical Heart Disease (Levine)	Saunders.
Heart Disease (White)	Macmillan.
Textbook of Medicine (Cecil)	Saunders.
Rheumatic Heart Disease (Coombs)	Wm. Wood and Co.

Section III

RHEUMATIC HEART DISEASE

	Paragraph
General	101
Acute rheumatic heart disease	102
Chronic rheumatic heart disease	103
Recommended texts	104

101. General.—All rheumatic involvement of the heart is the result of rheumatic fever whether that rheumatic fever was acute and typical or whether it was subacute and abortive in its manifestations. For convenience in discussion, rheumatic heart disease is grouped into two divisions, the acute type occurring during the acute rheumatic fever and the chronic type, which is the end result of damage incurred during the acute phase. It is to be borne in mind that the two types are overlapping, frequently superimposed, and that the chronic results cannot occur without preceding acute involvement.

102. Acute rheumatic heart disease.—*a. General.*—(1) The various manifestations of acute rheumatic heart disease are a definite part of the picture of acute rheumatic fever, occurring with and often prolonging the symptoms and signs seen in the acute involvement.

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Often the carditis exists when there are no signs of the systemic disease other than those mentioned in section II as atypical, or there may be no indications whatsoever that the rheumatic fever exists. Conversely, it is fairly safe to say that the heart does not escape damage during any attack of the rheumatic fever group, although symptoms and signs of this involvement may be missing at the time of the acute episode. White states that "It is probable that in every case of rheumatic infection there is some heart disease, however, slight or transient, and that in a certain percentage of the total number there is a complete recovery with return to normal, or at least not sufficient deformity of valves or lesion of myocardium or pericardium to produce abnormal signs."

(2) The age of onset of cardiac involvement corresponds roughly with the age of onset in acute rheumatic fever. The acute carditis is rarely seen before the age of five, and is rather uncommon after the age of twenty. Females are slightly more subject to this disease than males.

b. Pathology.—(1) The invasion of the heart often involves all three structural layers, creating a rheumatic pancarditis. More commonly, however, the endocardium alone is involved, or the endocardium and myocardium are involved together. The pericardium is never affected without simultaneous involvement of the other two structures. It is interesting to note in passing that rheumatic heart disease is probably the most common cause of any pancarditis.

(2) Acute involvement of the endocardium is basically inflammation and edema, with a special type of end result. The inflammation centers about the valves and, if severe enough, extends from the valve surfaces to the adjacent mural endocardium. The mitral valve is most often involved, the aortic valve running a poor second in frequency. The tricuspid valve is rarely affected and the pulmonary valve practically never. The mitral and aortic valves are very commonly attacked simultaneously. The process develops as the inflamed and swollen valves become, as a result of the valvular movements, traumatized at their line of closure. At this line of trauma, erosion and minute ulcerations occur. If the process becomes very extensive, the ulcerative lesions spread along the valve surface and involve the adjacent heart wall and the chordae tendineae of the auriculoventricular valves. Over these erosions small sterile thrombi form, 1 millimeter or less in size, usually laid down in parallel rows along the line of closure (auricular surface of mitral valve and ventricular surface of aortic). As the process continues, these thrombi become flattened and heal by fibrosis. The healing thrombi or vege-

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tations become covered with endothelium as the process reaches its conclusion. The end result of the process of inflammation and healing, fibrosis and scar tissue contraction, produces deformity and thickening of the valve edges, the degree of such deformity depending entirely upon the extent and severity of the inflammation. Thus is laid the foundation (contracture deformity) for chronic valvular disease.

(3) Acute involvement of the myocardium is also an inflammatory process. This acute inflammatory process consists chiefly of minute areas of tissue necrosis and hemorrhage, the repair of which occasions the appearance of Aschoff bodies. Aschoff bodies are now taken as evidence of the healing process and are considered the typical pathological manifestation of rheumatic fever, comparable to the tubercle in tuberculosis. The lesion is a perivascular nodule, seen chiefly around the smaller vessels, consisting of groups of mononuclear and giant cells. These bodies vary in number with the severity of the involvement and leave practically no trace of themselves. Healing is by a very minute scar. If the involvement is extensive and severe, a recognizable diffuse fibrosis of the myocardium is the end result. Also, if the inflammation is severe and extensive, it is apt to lead to congestive failure and dilatation during the acute stages.

(4) The pericardial involvement is chiefly a serofibrinous process in which a large effusion is the exception rather than the rule. Rheumatic pericarditis is characterized by the completeness of its healing and the lack of obvious end results, pericardial adhesions being somewhat uncommon. Extensive adhesions do occur, but only as a result of the very severe involvements. In fact, both the myocarditis and the pericarditis are notable for the absence of obvious residuals except in the very severe lesions, whereas in the endocardium even the milder degrees of pathology leave definite deformity of some degree.

c. Symptoms.—(1) The symptoms of acute rheumatic carditis are usually very vague, and apt to be masked by the symptoms of the systemic involvement. In fact, it is often very difficult to tell definitely whether or not the heart is involved, inasmuch as the carditis is frequently mild. However, all degrees of carditis exist from the mildest to the very worst, in which the condition may even be fulminating, capable of producing death during the first or second attack. The safest attitude to accept, as emphasized previously, is that each attack is leaving damage or adding damage to that already imposed by previous attacks.

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(2) There are three symptoms that should be carried in mind when searching for rheumatic carditis: precordial ache, palpitation, and dyspnoea. The precordial ache is far the most characteristic and is usually dull, mild, and prolonged. It is possible, although rare, that this ache may be an actual pain, even to anginal severity, which so often accompanies the advanced aortic valve involvements. Palpitation is due to the rapid heart rate and extrasystoles, and can be very disturbing. Dyspnoea, when noted, is usually mild and due mostly to effort syndrome, rather than to congestive failure. In the fulminating cases, the symptoms are those of rapid and severe congestive failure, with its multitude of manifestations and complications.

d. Signs.—(1) There are many more signs of acute rheumatic carditis than there are symptoms. Those described are typical of an acute primary involvement; but it must be borne in mind that, in the second, third, or following rheumatic insults to the heart, the signs are more than apt to be superimposed on those of the damage left by the previous attacks. There are six signs of importance: fever, tachycardia, leucocytosis, systolic murmur, conduction disturbances, and pericardial friction rub.

(2) Fever is usually low grade, prolonged, and very significant. Persistence of fever after subsidence of acute articular symptoms is to be regarded as definite evidence of cardiac involvement. Tachycardia is usually rapid, prolonged, and much more rapid than the amount of fever warrants. Persistence of tachycardia after subsidence of acute symptoms is to be regarded as of the same significance as fever. Leucocytosis is usually of low grade and persists long after the disappearance of acute symptoms. The three together, fever, tachycardia, and leucocytosis, constitute a syndrome absolutely diagnostic of rheumatic carditis when following acute rheumatic fever. A soft, blowing, systolic murmur at the apex, or its predecessor, roughening or prolongation of the mitral first sound, occurring during or after acute rheumatic fever, can also be taken as definite evidence of cardiac pathology. Likewise, conduction disturbances as shown in the electrocardiogram, a prolonged PR interval, or dropped beats are to be taken as definite evidences of cardiac involvement. Premature supraventricular beats are indicative, although not as definite, as the other two EKG signs. A pericardial friction rub occurs only in the more severe cases and is the only absolute evidence of involvement of the pericardium in the absence of extensive effusion.

(3) Other signs seen in the advanced cases due to repeated rheumatic involvement, or in the fulminating cases, are those of congestive failure: dyspnoea, lung congestion, and peripheral edema. This con-

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gestive failure usually is of the early, mild type, easily recovered from except in those rapidly fatal malignant cases. Enlargement of the heart, usually dilatation, may also be present and detected by physical signs or X-ray. Marked dilatation will have both apical and aortic murmurs. Blood pressure may be normal or low. Children may suffer a slight secondary anemia of prolonged duration, which is suggestive but not pathognomonic.

e. Prognosis.—The prognosis as to recovery for each individual attack is normally good. Single attacks are usually survived, although as previously noted, death occasionally occurs during a single attack. It should be remembered that following recovery from each attack, whether typical or atypical, some definite damage has taken place and some degree of scarring is present, even though no residual symptoms make these facts apparent. In fact, although there may be some signs in more advanced cases, there will be no symptoms as long as the cardiac reserve is of sufficient degree to take care of the damage. In general, the younger the patient with cardiac damage, the more serious it is apt to be and the poorer the prognosis for length of life. Likewise, the more recurrences the more serious the heart damage is apt to be and the poorer the prognosis for longevity. Few people with rheumatic heart damage greater than that which can be classified as mild will live much beyond the age of fifty. At best, a child who has suffered one or more acute exacerbations of rheumatic carditis can expect to arrive at adulthood with some degree of heart damage. Most of this heart damage will be severe enough to be classified as chronic rheumatic heart disease.

f. Treatment.—Treatment of acute rheumatic carditis is essentially a prolongation of the treatment of acute rheumatic fever. Bed rest, good nursing care, adequate diet, and symptomatic medication constitute the main measures to be taken. Rest in bed is to be continued until the temperature no longer rises above 99, the white blood count remains below 9,000, the pulse below 100, the signs and symptoms of infection have disappeared, and the nutrition improved. The necessity of good nursing care needs no comment. Diet should be of the high vitamin type, of high caloric content, and easily digestible. A warm, equitable climate seems to hasten recovery, and convalescence in such a climate is to be very highly recommended. The recent literature has contained several reports of institutions established in Florida and elsewhere for the convalescent care of rheumatic fever and carditis. In all reports the results seem to be very gratifying.

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103. Chronic rheumatic heart disease.—*a.* Chronic rheumatic heart disease is due entirely to the residual pathology left in the heart by one or more attacks of acute rheumatic carditis. This residual pathology is the valvular deformity occasioned by the cicatricial contraction of the valvular tissue in the healing of the inflammatory process. Therefore, in practically every case, chronic valvular disease of some degree of severity is present. Pericarditis, chiefly in the form of adhesion, less often in the form of constrictive pathology, occurs in less than 10 percent of cases, most often in conjunction with the valvular lesions. Permanent myocardial lesions due to the scar tissue healing of the muscular inflammation occur rather rarely. This condition is manifested chiefly by blocks of various degrees.

b. Chronic valvular disease is by far the most important of the manifestations. The mitral valve alone is involved in roughly 60 percent of cases, the aortic valve alone in 5 percent of cases, and the mitral and aortic valves in combination in 35 percent. The tricuspid and pulmonary valves rarely show chronic residual deformity; the former infrequently, the latter practically never. For the signs and symptoms of chronic valvular disease see paragraphs 52 to 57. Pericarditis and blocks are likewise discussed in some detail in paragraphs 118 to 120 and 196 to 200, respectively.

c. It is to be emphasized that chronic valvular disease is just one phase in the progress of chronic rheumatic heart disease. The entire affliction, from the point of manifest mitral stenosis to ultimate death, is one of a relentless progression, the tempo of which is determined by both intrinsic and extrinsic factors. The intrinsic factors are chiefly those of the degree and extent of the valvular deformity. The extrinsic factors are those concerned with the load placed on the heart by the pace and type of living indulged in by the individual, plus the coexistence of other diseases. As long as the cardiac reserve is able to take care of the bodily demands, plus the extra load occasioned by the valvular deficiency, the individual will lead a fairly normal life. When the cardiac reserve is no longer able to carry that combined load, the process leading to the ultimate end becomes manifest.

d. The next step in the progression following the chronic valvular disease is dilatation and enlargement of the heart as an attempt to carry the extra load. Congestive failure eventually follows. Death ultimately occurs. This process may be a matter of 1 or 2 years, or extend over a period of 1 or 2 decades. It is commonly marked by many remissions, but in any case, once the reserve has broken, the

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individual becomes a cardiac cripple of gradually increasing degree. Congestive failure occurs in well over one-half of the cases of chronic rheumatic heart disease.

e. The progression from stenosis through enlargement to congestive failure and death is fairly classical, and is to be considered the predicted course in any case, unless interrupted by accident or intercurrent infection. However, there are two exceptions to this rule, auricular fibrillation and subacute bacterial endocarditis, both closely interwoven with congestive failure. Fibrillation, either alone or in conjunction with congestive failure as cause or result, occurs very commonly in the patients with marked mitral stenosis. Here, it is a rather common cause of death. Subacute bacterial endocarditis occurs in a small percentage of cases. It is one of the dangers faced by all adults with chronic rheumatic heart disease and it is emphasized in paragraph 107 that rheumatic valvular damage is one of the largest causes of subacute bacterial endocarditis.

f. The prognosis varies with the severity of the sequallae previously mentioned. Those who have slight lesions not sufficient to embarrass the cardiac reserve to any appreciable extent may live to old age. However, practically all of those definitely involved in the rheumatic progression will have died before the age of fifty, death commonly occurring between the ages of fifteen and fifty. All those in the advanced stages of chronic rheumatic heart disease will be cardiac cripples of varying degrees prior to death, the length of the invalidism depending upon the seriousness and rapidity of the process. Periods of invalidism as long as ten to twenty years are common, although the average is considerably less. All persons with chronic rheumatic heart disease are very susceptible to intercurrent infection and these infections are frequently the mode of termination of the case.

g. Treatment consists of symptomatic relief and a cardiac regime which will enable the patient to live within the limits of his reserve. Adequate rest, avoidance of overexertion and infections, especially the respiratory diseases, are to be emphasized. Further than this, little can be done other than to make the patient comfortable and take care of such exigencies as may arise.

104. Recommended texts:

Clinical Heart Disease (Levine)---------- Saunders.
Heart Disease (White)---------- Macmillan.
Diseases of the Heart (Lewis)---------- Macmillan.
Rheumatic Heart Disease (Coombs)---------- Wm. Wood and Co.

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SECTION IV

ENDOCARDITIS

Paragraph
General 105
Acute bacterial endocarditis 106
Subacute bacterial endocarditis 107
Recommended texts 108

105. General.—*a.* Endocarditis literally interpreted means an inflammation of the lining membrane of the heart. This inflammation is the forerunner of practically all valvular heart disease, the only notable exceptions being congenital conditions, arteriosclerosis, and syphilis. The condition is commonly classified into two types, bacterial and nonbacterial; the bacterial type being further classified into subacute and acute. The nonbacterial type includes that endocarditis occurring in rheumatic fever described in paragraphs 101 to 103, and the mild, silent, terminal type found frequently at autopsy as a complication of certain acute infections. It is possible that this so-called terminal type also exists in nonfatal severe diseases and that recovery does occur without its existence becoming known. However, being relatively unimportant, its further discussion will be omitted and the student is referred to the standard texts for details. In passing, the very rare occurrence of endocarditis with tuberculosis, lues, and actinomycosis should be mentioned.

b. Acute, subacute, and rheumatic types of endocarditis are very closely related. This is especially true in respect to pathology, severity, duration, and prognosis. All three entities have valvular vegetations as the basic manifestation. The vegetations in rheumatic carditis are of pinpoint size, those in the subacute type are larger, and those in the acute type are largest, often reaching the size of a pea. In severity, the same relationship holds true. Rheumatic carditis, except in the fulminating cases, is usually moderately severe, the subacute is more severe, and the acute type is always overwhelming. The reverse order is true in regard to duration and prognosis. In rheumatic carditis the individual attacks are a matter of weeks or months, although they may recur over a period of years. In subacute bacterial endocarditis the attack is a matter of months to a year, whereas in acute endocarditis it is a matter of a few days or weeks. Rheumatic endocarditis usually presages recovery from the individual acute attack, the subacute bacterial is fatal after a lingering illness, and the acute is rapidly fatal.

106. Acute bacterial endocarditis.—Acute bacterial endocarditis is an acute bacterial invasion of the endocardium, always sec-

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ondary to some other disease process and a part of an overwhelming blood stream infection. Many of its features resemble those of subacute bacterial endocarditis, but its course is much more rapidly fatal.

a. Etiology.—The condition is relatively uncommon, occurring at any age, and with slightly greater frequency in males. There are no other etiological factors of importance such as climate, geographical distribution, or environment. The organisms responsible for this condition are chiefly cocci, the most common of which are streptococcus hemolyticus, staphylococcus aureus, pneumococcus, gonococcus, and meningococcus. The colon bacillus and the influenza bacillus are the other common offenders. Many other bacteria have been named as causative factors in isolated cases, and practically all of the common pathogenic organisms have been reported at one time or another. The organisms gain entrance into the blood stream during the acute illness and settle upon the endocardium, commonly selecting the site of previous damage. However, it is almost equally as common for the lesion to occur on previously undamaged valves.

b. Pathology.—(1) The basic pathology is inflammation of the endocardium which leads to the formation of thrombi at the points of injury or trauma. The whole process is very similar to that noted in rheumatic carditis, although much more severe and rapid. The vegetations are thrombi, made up of fibrin, leukocytes, and living bacteria and often become 1 centimeter or more in size. They involve the valve margins, extend down the valve surfaces to the neighboring walls and chordae tendineae, and frequently get into the aorta. Unlike subacute and rheumatic types, all valves are individually subject to involvement, the left side of the heart being involved somewhat more frequently. The mitral and aortic valves are attacked with about equal frequency, the tricuspid next, and the pulmonary valve least often. This rather unusual incidence of involvement is explained by the fact that acute bacterial endocarditis, unlike the subacute type, attacks undamaged valves as well as those already deformed.

(2) As the disease progresses, ulceration occurs at the site of vegetations. This often results in perforation or aneurysm of the valve cusps, rupture of chordae tendineae, or mycotic aneurysms in surrounding structures. A more common accompaniment of the advanced process is the snapping off of thrombi into the blood stream to produce embolic phenomena. Any of these occurrences may be the terminal events in the disease.

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c. Symptoms.—The symptoms are those of an acute septicemia, superimposed on the manifestations of the causative infection. Chills, sweating, prostration, and delirium are especially common. Symptoms relative to the heart are usually missing or, if present, are overshadowed by the severity of the septicemia. Later in the course of the disease the symptoms occasioned by embolism become most noticeable. Emboli lodge most frequently in the brain, viscera, lungs, extremities, and heart and produce symptoms more or less characteristic of the organ involved.

d. Signs.—(1) At first the signs are not at all typical, being chiefly those of the septicemia. Prior to embolic phenomena, the signs of septicemia and the causative condition mask all the manifestations of the endocarditis. The diagnosis is an unusually hard one to make for this reason, and the condition is frequently missed until late in its course or until autopsy.

(2) Suggestive signs are a leukocytosis of 20,000 or 30,000, slight anemia, slight splenic involvement, positive blood culture, and acute nephritis. However, all of these are also signs of a severe infection and can in no way be construed as being exclusively cardiac in origin. The diagnosis will usually not be made until petechia appear, the signs of embolism occur, or shifting, changing types of heart murmurs become evident. These three signs occurring in a severe fulminating infection make the diagnosis of endocarditis almost positive. Blood cultures cannot be said to be any more than confirmatory. EKG is not at all helpful, the abnormalities seen being often only a prolongation of the PR interval, and this only in some cases.

e. Prognosis.—The disease, which lasts from several days to 3 or 4 weeks, progresses rapidly to a fatal termination. Death is due primarily to toxemia and exhaustion, although it sometimes occurs as a result of either congestive failure or embolism. Recovery is very, very rare, and has been reported in the literature only on one or two occasions.

f. Treatment.—There is no specific treatment for this condition. Practically everything has been tried; all drugs, serums, and procedures ending in failure. Transfusions are of some temporary benefit, but the best that can be said is that they are merely helpful. In cases where there exists a specific serum for the causative organism, such as the meningococcus, the serum should at least be tried. Symptomatic relief, supportive treatment, and good nursing care are practically the extent of therapeutic measures to be offered.

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107. Subacute bacterial endocarditis.—Subacute bacterial endocarditis, like the acute type is an invasion of the endocardium by bacteria. It is a fatal, lingering illness, very similar to the acute type in many of its manifestations.

a. Etiology.—This condition is more common than the acute, occurs at any age, and has the highest incidence between the ages of twenty and forty. Males seem more susceptible than females. Exposure, accident, or strain may precipitate an attack, although they are not causative factors. Climate, geographical distribution, and environment have no bearing on the subject except insofar as they relate to the incidence of rheumatic fever. One organism, streptococcus viridans, is the cause of approximately 95 percent of the disease, the gonococcus and influenza bacilli accounting for the remainder. Unlike the acute type, the condition attacks only those valves that have been previously damaged. Streptococcus viridans is believed to circulate in the blood stream at times and to invade the damaged valvular endocardium when the general resistance is low. Rheumatic valvular deformity accounts for over 80 percent of cases, the congenital defects account for the majority of the remainder. Levine states that from 20 to 25 percent of all cases with valvular heart disease die of bacterial endocarditis, either acute or subacute. Another peculiarity of the disease is that it attacks chiefly those in whom the valvular disease is well compensated and who are in apparently good health rather than those with failing reserve or fibrillation.

b. Pathology.—(1) Here, as in the acute type, the basic pathology is inflammation of the endocardium, which results in vegetations being formed on the affected valves at the points of previous damage. These vegetations are irregular masses of thrombus formation containing fibrin, blood cells, and bacteria. They are smaller than those seen in the acute type and tend to become more flattened in contour. They also spread from the line of closure over the entire valve surface down onto the chordae tendineae and onto the neighboring mural endocardium. The pathological process here is much slower than in the acute endocarditis, although it leads to practically the same end results: ulceration, perforation, aneurysm of the valves, mycotic aneurysms, and small contact or extension muscle abscesses. These abscesses may ultimately perforate the septum or even the myocardial wall. Infrequently they damage the bundle of His or one of its branches to a sufficient extent to cause varying degrees of heart block.

(2) Unlike the acute type, the left side of the heart is much more frequently involved than the right, and the mitral valve is the seat

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of pathology more than the aortic. This fact is explained by the incidence of valvular damage following rheumatic fever. When the right side is involved, the valves or structures attacked are usually those deformed by congenital heart disease. The pulmonary valve is rarely involved. Bicuspid aortic valves seem especially prone to attack.

c. Symptoms.—The symptoms of subacute bacterial endocarditis are those of a low grade, lingering infection, much milder in its early manifestations than the acute type. As typical of infections of low virulence, the onset is insidious, often being initiated by some otherwise unimportant event, such as simple respiratory infections, childbirth, minor surgery, minor accidents, or secondary infections. Fatigue, afternoon fever, listlessness, loss of weight and appetite, malaise, and anorexia are common. Later, chills, fever, prostration, and loss of strength all appear in gradually increasing severity. The symptoms occasioned by embolism appear in the terminal stages and often dominate the picture from that point on. Usually there are no symptoms directly referable to the heart.

d. Signs.—(1) The signs of the subacute condition are much more numerous and definite than the symptoms. Eleven signs of importance must be listed: fever, anemia, leukocytosis, petechiae, purpura, splenomegaly, clubbing of fingers, valvular murmurs, embolism, renal involvement, and positive blood cultures. The fever is at first low grade, apt to be missed by casual check, and occurs in the afternoon and evening. As the course of the disease progresses, the fever becomes much more marked, remitting in type, often having wide daily swings from normal in the morning to the higher degrees in the evening. The anemia is a true secondary anemia, often of considerable degree, becoming progressively worse and resulting in a characteristic pallor known as "cafe au lait." The leukocyte count ranges between 10,000 and 15,000, rarely above the latter figure. Petechiae appear fairly early in the course of the disease, and are not embolic phenomena but are due to blood vessel damage. They come and go, are of fairly generalized distribution, and are very typical of the condition. In the fingernails, conjunctiva, and mucous membranes, they assume the "splinter" shape, otherwise they are characteristically round. Detection is made earliest in the sclera or in the hand and forearm. Probably the best way to bring out latent petechia is to pump up a blood pressure band on the arm and leave the constriction in place until cyanosis is evident. If petechiae are at all present, this will bring them out into startling clearness. Pupura is less commonly seen than petechia, but when present it is frequently severe.

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Splenomegaly is a constant sign, often appearing early, and the spleen becomes larger and more tender as the disease progresses. Clubbing of the fingers also appears early in the course of the disease, and when it can be determined that it was not present prior to onset this sign constitutes a very valuable diagnostic aid. The murmurs of valvular heart disease will always be present after the earliest stages. Even though the preexisting valvular damage produced no signs prior to the subacute endocarditis, the added damage is usually severe enough to cause marked signs and even enlargement. This fact is of such importance that a diagnosis of subacute bacterial endocarditis cannot be made in the absence of murmurs. Embolism, as in acute endocarditis, appears in the terminal phases of the subacute disease and causes a multiplicity of signs, all of which depend upon the size and location of the vessels occluded. The spleen, kidneys, limbs, and brain are the sites of most frequent involvement, the lungs being hit when the seat of pathology is in the right heart. Further complicating the embolic phase is the fact that most of the emboli are infected and set up local pyogenic abscesses at the site of lodgment. Renal involvement is early, usually passing through the stage of mild, acute nephritis to a chronic glomerular type, later again becoming acute with the lodgment of emboli. Positive blood cultures in the presence of two or more of the above signs help make the diagnosis. However, a positive culture is very hard to obtain and special technique is required. Diagnosis is to be made on a combination of any four or five of the above signs. The most important, at least three of which should be present (except when blood culture is positive) before making a diagnosis, are embolism, murmurs, clubbing, splenomegaly, and petechiae.

(2) One of the newer methods of confirming a diagnosis of subacute bacterial endocarditis, and one of some importance, is the local reaction to intradermal injections of killed streptococcus viridans vaccine. This reaction is negative in subacute involvement and distinctively positive with varying sized wheals in rheumatic fever or tuberculosis. This reaction seems to be fairly dependable in a large majority of cases.

e. Prognosis.—This disease is characterized by slow progress, gradual increase in severity of symptoms and signs, and a very definite downhill course. The duration varies from a few months to a year or more, cases having been reported which lasted as long as 2 years. The average duration is 6 months. Death is due to either toxemia, congestive failure, embolism, intercurrent infections, or uremia. Toxemia with exhaustion is the most common cause. Recovery is very

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rare, possibly occurring in the milder cases, but never in a clear-cut case. Diagnosis is often missed prior to appearance of petechia or clubbing; tuberculosis, rheumatic carditis, and undulant fever being most often confused with it.

f. Treatment.—There is no specific treatment for this condition. Here again practically everything in the way of therapy has been tried, all with universal failure. Symptomatic relief, supportive treatment, and good nursing care are about all that can be offered. Transfusions may be helpful in that they frequently give temporary relief. The effects of sulfapyridine and heparin on the condition are in the process of investigation. It is yet too early to report definite results, although the principle of this type of therapy offers some promise.

108. Recommended texts:

Heart Disease (White)	Macmillan.
Clinical Heart Disease (Levine)	Saunders.
Diseases of the Heart (Lewis)	Macmillan.

Section V

ACUTE TOXIC MYOCARDITIS

	Paragraph
General	109
Etiology	110
Pathology	111
Symptoms	112
Signs	113
Prognosis	114
Treatment	115
Focal infections	116
Recommended text	117

109. General.—Acute toxic myocarditis, as such, is of little importance to us as examining flight surgeons because of two factors; first, death either occurs during the acute illness or, second, recovery when it occurs is usually complete from a cardiac standpoint. The toxic involvement of the myocardium in the acute fevers and sepsis rarely produces serious residual heart disease. Also, it is more or less of an axiom that a previously healthy heart rarely gives way under acute infection except in two diseases, rheumatic fever and diphtheria.

110. Etiology.—It is now thought that many more of the acute illnesses have coincident toxemic changes in the heart muscle than was formerly believed. The diseases which commonly have this myocardial involvement are rheumatic fever, diphtheria, scarlet

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fever, typhoid fever, pneumonia, influenza, septic infections, tuberculosis, and yellow fever. Trichiniasis also produces degenerative myocardial changes around the rather infrequent trichina heart lesions. Acute diseases other than those mentioned very rarely, if ever, involve the heart.

111. Pathology.—*a.* The pathological changes produced in a majority of the above-mentioned illnesses are due to the action of bacterial toxins rather than to direct invasion of the musculature by bacteria themselves. This toxic action produces almost universally an inflammation, with cloudy swelling and coincident changes, which is followed by degeneration and often necrosis of the individual muscle fibers. The degeneration seen is often accompanied by multiple minute hemorrhages into the muscle, seen in greatest numbers subendocardially and beneath the pericardium. When recovery occurs, the healing process is remarkably complete, residual scarring being present, but very difficult to determine at autopsy.

b. There are several exceptions among the acute illnesses which do not show the typical toxic degenerative processes noted above. Tuberculosis of the myocardium is characterized by the presence of tubercles in the muscle structure, which involvement occurs only as a part of the acute miliary lung disease. The myocarditis due to trichiniasis has been noted as being due to the presence of the parasite in the muscle tissue. Septic infections sometimes produce minute abscesses throughout the musculature, such invasion usually being a minor portion of the picture of an acute septic pancarditis. Scarlet fever is thought to precipitate latent rheumatic lesions both in the endocardium and myocardium. At best, there is a rather high rate of arthritic and cardiac involvement noted as sequellae to this disease, and most authorities believe that the process is rheumatic rather than due entirely to scarlet fever.

112. Symptoms.—Acute myocardial involvement usually shows little in the way of symptoms. When symptoms are present, they usually consist of the triad, precordial discomfort, dyspnoea, and palpitation. The precordial discomfort is frequently a sense of oppression or constriction rather than actual pain. A sense of weakness is a fourth finding, prolonged weakness being especially a result of influenza and thought by many to be due to results of the myocardial involvement. This conjecture is under dispute at the present time inasmuch as there are usually no remaining cardiac signs in most of these cases.

113. Signs.—Signs are likewise rather infrequent prior to a very acute and serious involvement. When present they consist chiefly

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of electrocardiograph changes as manifested by blocks of varying degrees, dilatation with its telltale murmurs, and tachycardia. Frequently, cyanosis or ashiness denotes the onset of circulatory collapse. The electrocardiographic changes as demonstrated by the conduction and T-wave deformities are perhaps the most reliable indication of myocardial involvement prior to the severe stages. Very few of these changes persist, even in minor degrees, following recovery.

114. Prognosis.—The prognosis as to recovery from acute myocardial involvement is, in general, good. Except in rheumatic fever and diphtheria, death is usually due to some group of circumstances other than cardiac. In diphtheria about one-third of the cases, untreated by serum, die of circulatory failure. Death is often very sudden and dramatic; the patient may be sitting up in bed in an apparently good convalescence and fall over dead. The presence of a majority of the above-given signs in acute diphtheria is of serious prognostic significance even though half of the cases presenting signs and symptoms do recover. The prognosis in rheumatic fever has already been discussed. In all except rheumatic fever and scarlet fever, if recovery is made, there is little or no residual damage, repair taking place more or less completely. Occasionally, there will be a persistent heart block where scarring has involved some of the conduction tissue of the heart; but other than this there are usually no clinical signs to indicate that the heart was ever involved.

115. Treatment.—Treatment is that of the acute illness with especial care being taken to prolong bed rest, nursing care, and supportive treatment until all the signs of infection have disappeared. In diphtheria, the early and adequate use of antitoxin will prevent most of the cardiac involvement; in fact, the use of antitoxin in recent years has reduced the diphtheritic myocardial involvement to almost negligible amounts.

116. Focal infections.—At this time focal infections should be mentioned. Focal infections of any type should be considered deleterious to the heart, often precipitating failure or arrhythmia in already existing heart disease, or precipitating bacterial endocarditis. Some observers even believe that focal infections are a cause of heart disease; but probably if one considers them more as an added burden to be carried by either a normal or diseased heart, he will be on much safer ground. Active focal infections often increase anginal symptoms and their removal or treatment will markedly improve prognosis in some cases. Focal infections will, on occasion, induce arrhythmias of various degrees, from extrasystoles to fibrillation, even in the absence of heart disease. Such arrhythmias are usually not of great

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importance unless prolonged, and usually respond immediately to removal of the cause.

117. Recommended text.

Heart Disease (White) ---------- Macmillan.

Section VI

PERICARDITIS

Paragraph
General ---------- 118
Acute pericarditis ---------- 119
Chronic pericarditis ---------- 120
Recommended texts ---------- 121

118. General.—Pericarditis is an inflammatory reaction involving the serous surfaces of the pericardium, secondary to some other underlying causative condition. The most common causes of pericarditis are rheumatic fever, pneumonia, tuberculosis, nephritis, and the myocardial infarction which is secondary to coronary thrombosis. The inflammation is most often sterile, is fairly common in occurrence, and is frequently undetected. It occurs in two types, acute and chronic. This division is more or less arbitrarily made for the purpose of classification and it is very difficult to determine the dividing point between the two. Pericarditis of either rheumatic or uremic origin is sometimes classified as subacute.

119. Acute pericarditis.—Acute pericarditis consists of four main types, fibrinous, serofibrinous, purulent, hemorrhagic. For a discussion of two other rarer types, malignant and traumatic, the student is referred to the larger texts. Certain other acute conditions arise in the pericardium which are not pericarditis, but which give many of the same symptoms, signs, and end results. These are three in number: hemopericardium, in which for some reason (trauma, ruptured aneurysm, etc.) frank hemorrhage occurs into the pericardial sac; hydropericardium which is a concomitant part of the picture of anasarca or terminal cachexia; and pneumopericardium in which air gets into the pericardial sac as a result of perforation of esophagus or bronchii, or as an incident to pneumothorax. Hydropericardium and hemopericardium embarrass heart action, the latter often acting as a tamponade, causing an acute constrictive pericarditis. For further discussion of these conditions, the student is again referred to the recommended texts.

a. Etiology.—(1) Acute or subacute pericarditis is always secondary to some other underlying disease condition. It is fairly common and, although frequently missed in the living, is seen in roughly 5

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percent of autopsies. It occurs most commonly in youth, and males predominate markedly in the disease incidence.

(2) The disease conditions underlying the fibrinous type of pericarditis are: rheumatic fever, infectious diseases, myocardial infarction, and uremia. In rheumatic fever the condition is usually a transient accompaniment of an acute pancarditis, and generally recedes with a minimal amount of damage. Of the infectious diseases, pneumonia, influenza, tonsillitis, tuberculosis, typhoid fever, and septic conditions are the most common offenders. Pneumonia and sepsis more frequently than not produce the purulent type, but occasionally do have fibrinous involvement only. Tuberculosis is always secondary to involvement of the pleura, lungs, or meadistinal glands. Typhoid fever, influenza, and tonsillitis usually cause a more or less symptomless type of pericarditis which very frequently escapes detection prior to autopsy. In myocardial infarction, the pericarditis is localized over the area of myocardium involved and then is present only when that process is extensive enough to reach the pericardial surface. Uremic pericarditis is a silent part of the picture of terminal nephritis and is very rarely detected in the living. Other disease entities have been reported at rather rare intervals as causing pericarditis, but for all practical purposes can be disregarded.

(3) The other three types have somewhat similar cause. The serofibrinous type of pericarditis has essentially the same causative factors as the fibrinous, except that the uremic and infarction types never produce serofibrinous exudate of appreciable degree. The purulent type has as its chief cause two diseases, pneumonia, and sepsis. This complication of pneumonia is somewhat rare, and practically always accompanies an empyema involving the left pleural sac. It occurs late in the course of the disease and is usually a part of the terminal picture. The pyogenic pericarditis of sepsis is either blood borne, due to extension of a neighboring process, or carried in by trauma, such as stab wounds. Neighboring processes which furnish the source for extension are empyema, an ulcerated esophagus, intrathoracic abscesses and the migration of pus from pyemic conditions below the diaphragm. In the hemorrhagic type, malignancy, either primary or metastatic, and tuberculosis are the chief offenders.

b. Pathology.—(1) In general, the fibrinous stage is the earliest lesion, serofibrinous the next, and purulent or hemorrhagic the last in the progression. In the fibrinous type, the serous surfaces first become injected, swollen, and incur deposits of lymph cells over the involved area. This lymph deposit develops into a layer of fibrinous tissue which varies in thickness up to 1 centimeter. The surfaces present a

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typical shaggy "bread and butter" appearance when separated, due to the soft, fibrinous deposit. The condition may be localized to a small area or be generalized and involve the entire pericardial sac. Either surface of the pericardium may be involved initially, but ultimately both opposing surfaces are usually affected equally. Healing is by fibrosis, and the end result varies from a slight thickening of the pericardium to dense adhesions with obliteration of the pericardial sac. In the most serious cases, extrapericardial adhesions sometimes arise between the parietal pericardium and neighboring structures. Except for death, this is the most serious end result possible. The serofibrinous type is always a sequel of the fibrinous condition, the effusion varying in amount up to 3,000 cubic centimeters. Quantities less than 300 cubic centimeters are not detectable. This outpouring of fluid separates the inflamed surfaces and distends the pericardial sac, sometimes to an astounding degree. Ordinarily, the parietal pericardium is extremely resistant to stretching, and the more rapid effusions and hemorrhages meet with a great resistance. The resultant intrapericardial pressure causes an acute embarrassment to the heart action, which, if not promptly relieved, is often fatal. On the other hand, the slowly increasing pressure of an average effusion encounters little difficulty in making room for itself. The result of this slow increase in size of the pericardium is chiefly that of pressure on surrounding structures. Healing here, except in the case of TBC, usually occurs spontaneously following the recession of fluid, and the end result is the same as in the fibrinous types.

(2) In the purulent type, the fluid in the pericardial sac is anything from a large number of pus cells in the effusion to a frank, thick, heavy pus. Usually, the quantity is fairly small. Death occurs in a short time from sepsis unless adequate surgical drainage is instituted. In the hemorrhagic type, blood cells are present in the effusion in varying numbers, not enough to be a frank hemorrhage. This type is usually a terminal state in either malignancy, tuberculosis, or a fulminating rheumatic pancarditis.

c. Symptoms.—(1) Symptoms are chiefly those of the underlying condition until an effusion occurs large enough to cause pressure. Very frequently the fibrinous pericarditis is silent, this being especially true of the uremic type. Huge areas of the visceral pericardium and much of the parietal surface can be involved without production of pain. However, when the pleural or diaphragmatic surfaces of the pericardium are involved, pain is the result. This pain may be sharp and severe, or it may be a dull, prolonged ache. Usually, it is centered over the precordium and referred either to the left shoulder or epigastrium.

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(2) When effusion becomes large enough to cause pressure symptoms, we have two types of manifestations, those due to pressure on the heart itself, and those due to pressure on neighboring structures. Pressure on the heart itself causes an insufficiency of blood flow into heart, lungs, and great vessels and results in all of the symptoms of acute failure, venous congestion, dyspnoea, enlargement of the liver, etc. Pressure on surrounding structures, especially lung and mediastinum, is manifested by symptoms arising from those structures such as dyspnoea, irritative cough, dysphagia, and hoarseness. Purulent pericarditis has the symptoms of effusion, plus those of sepsis. The hemorrhagic type has only the symptoms of effusion, plus those of the underlying cause.

d. Signs.—(1) Fibrinous pericarditis has only one sign which is characteristic of the condition, the friction rub. This sign is present in less than half of the cases, and when present is usually transient and inconstant. The typical friction rub is a loud, coarse, rough, grating to and fro murmur heard close under the chest wall, which is increased in intensity by pressure of the stethoscope or by leaning the patient forward. It is best heard in the third or fourth left interspace at the sternal border. At times it is also heard over the entire precordium. The friction rub usually disappears when the inflamed surfaces of the precordium become separated by the appearance of effusion, or when fibrous healing occurs. Any variation in the characteristics of the friction rub is possible, such variations often leading to confusion with valvular murmurs.

(2) Pericardial effusion, on the other hand, has a number of signs more or less characteristic of the disease. The most important of these are enlargement, increase in venous pressure, low systolic blood pressure, narrowed pulse pressure, paradoxical pulse, changes in heart sounds, changes in PMI, and Ewart's sign. Enlargement is detected either by percussion or X-ray. The area of dullness will usually extend well into the left axilla and well beyond the right sternal border. Here the PMI is usually well within the left border of dullness, unless there is an accompanying dilatation or hypertrophy. The X-ray shadow will be globular in the reclining position and pear-shaped in the upright position. This change of shape to X-ray is one of the most reliable signs in distinguishing effusion from true hypertrophy. Increase in venous pressure results from embarrassment of the heart action due to pressure of the fluid on the heart itself. The signs resulting are those of congestive failure, with enlargement of the liver and ascites being especially prominent. The systolic blood pressure is usually low and the pulse pressure

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narrows appreciably. The paradoxical pulse is a waxing and waning of the pulse with breathing, the pulse becoming weaker or disappearing with inspiration and returning to near normal on expiration. Heart sounds diminish in intensity and quality and often become muffled and distant. The PMI tends to become diffuse, or it may disappear entirely. Ewart's sign, which is due to the accumulation of fluid in the posterior pericardial sac and resultant pressure on lung tissue, appears at the angle of the left scapula. Here, the compression of the lung causes dullness, bronchial breathing, and increased fremitus to appear in a small area. This finding is often mistaken for pneumonia.

(3) The electrocardiograph pattern of acute pericarditis is fairly definite and characteristic. The pattern is too extensive to be listed at this time and the student is referred to the literature for details.

(4) The larger effusions are due to tuberculosis or rheumatic fever, the tuberculous effusions characteristically being very large and troublesome. Any large, sterile, persistent effusion or one which has a tendency to recur or reaccumulate should be cause for immediate suspicion of that disease. Rheumatic effusions usually subside spontaneously and rarely reach proportions which render tapping necessary. Guinea-pig inoculation of the effusion makes the diagnosis of the tuberculous type easy and absolute.

e. Prognosis.—The prognosis of acute pericarditis varies with the type and cause. In general, fibrinous pericarditis carries a hopeful outlook, usually subsiding spontaneously with the recession of the causative infection. However, two other results are possible, the development of effusion or, as in the case of uremia, death from the underlying cause within a short time. The development of pericarditis in uremia usually indicates that death will occur within a month. In the serofibrinous type, most of the trouble arises from the pressure symptoms, and death may occur from the marked embarrassment of the heart action. Tuberculous pericarditis is most often ultimately fatal, death usually being the result of the pericarditis, plus a combination of other factors characteristic of the disease. Rheumatic effusion usually subsides with the acute attack of rheumatic carditis unless that attack is of the fulminating type. Purulent pericarditis is always fatal unless early and adequate surgical drainage is established and the patient possesses a remarkable degree of resistance. In the hemorrhagic type the prognosis is that of the underlying cause, pericarditis being merely a complication which may hasten the end somewhat.

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f. Treatment.—Treatment of acute pericarditis is basically that of the underlying condition. In addition to this, symptomatic relief, good nursing care, and bed rest until the acute condition subsides are indicated as in all other heart affections. Pain and aching can sometimes be handled by such simple expedients as an ice cap to precordium, heat, or counterirritants. More often narcotics will be required, although salicylates given in the rheumatic type are very helpful. Removal of excess fluid in the large effusions relieves pressure symptoms and should be done whenever the signs of the pressure develop. The student is referred to the larger heart texts for the technique of this procedure. Adequate surgical drainage is the only possible treatment for purulent pericarditis and the earlier it is carried out, the more chance the patient has of recovery. Whenever purulent type is suspected, exploration should be done to determine its presence, using either a needle or trocar for the purpose, as indicated by the thickness of the pus encountered.

120. Chronic pericarditis.—Chronic pericarditis is that condition in which the pericardium becomes fibrous, thickened, and, in the more advanced degrees, adherent between the two layers. The degree of disability produced depends entirely upon the amount of embarrassment caused in the heart by the adhesions which obliterate the sac and, in many instances, firmly fix the heart to the surrounding tissues and structures.

a. Etiology.—Chronic pericarditis is considered by most authorities to be the end result of an acute inflammation of the pericardium. Like the concept of chronic rheumatic valvular disease, this is held to be true whether the acute condition was evident or not, and the finding of chronic pericarditis is to be taken as prima facie evidence that the acute condition existed at some time in the past. Rheumatic fever, pulmonary and pleural disease, myocardial infarction, and hemopericardium are the disease entities which cause the condition most frequently. Tuberculosis occasionally leaves chronic pericarditis as an end result. Many other diseases are given as cause, but much less frequently than those listed. The condition can occur at any age, the peak of incidence being between thirty and thirty-six.

b. Pathology.—The healing process which terminates the nonfatal acute pericarditis results in scarring and thickening of one or both layers of the pericardium. This fibrosis varies from small, irregular patches involving only one layer to the complete obliteration of the pericardial sac. This latter condition is known as concretio cordis. All graduations exist between these two extremes, both as to

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thickness of the fibrous layers and as to the extent of the areas involved. In the extreme degrees, areas of calcification are often found in this fibrous tissue. Concretio cordis may exist either alone or with its complication, chronic mediastinopericarditis, in which the parietal pericardium is bound in various degrees to the neighboring structures. Concretio cordis in the uncomplicated state is of importance only in the degree to which it constricts the heart itself and embarrasses its action. The adhesive process in chronic mediastinopericarditis may firmly anchor the parietal pericardium to the chest wall, mediastinum, or diaphragm, which results in a syndrome known as chronic constrictive pericarditis. This fixation of the heart results in cardiac compression and constriction of the great vessels, causing the heart to overwork markedly against the added load (pulling against the structures adhered to), and to become enlarged. Constriction of the superior vena cava results in a condition known as the superior mediastinal syndrome, in which there are evidences of venous congestion in the head and upper extremities. Constriction of the inferior vena cava, which is much more common, and known as Pick's disease, results in a condition known as the inferior mediastinal syndrome. Pick's disease results in a pseudocirrhosis of the liver, and compression of the hepatic veins, producing evidences of venous congestion in the abdomen and lower extremities. The right heart is usually more constricted than the left.

c. Symptoms.—The presence or absence of symptoms in this condition is entirely dependent upon the amount of constriction imposed upon the heart. In the greater percentage of cases symptoms are entirely absent and the disease is first discovered at autopsy. In the more serious degrees, the heart wears itself out pulling against the extra load, and the symptoms denoting the various degrees of congestive failure soon appear. In the mediastinal syndromes the symptoms of venous congestion are in the areas mentioned, of which the discomfort produced by the ascites is the most marked. Dyspnoea and progressive weakness are two other outstanding symptoms which should be noted as present in most advanced cases, both of which are part of venous congestion.

d. Signs.—(1) Signs like symptoms are dependent entirely upon the amount of constriction placed upon the heart and great vessels. Here again many of the milder conditions produce no signs whatsoever, the reserve of the heart being able to carry the slight additional burden without any manifestations. In simple concretio cordis there will be few if any signs prior to the onset of congestive failure. En-

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largement may be noted and there may be calcification of the pericardium detected in the X-ray.

(2) In chronic mediastinopericarditis there are several signs, all dependent upon either fixation of the heart or upon constriction of the heart and great vessels. Fixation of the heart is manifested by lack of shift of its position and by systolic retractions of the chest wall. The normal heart shifts from 2 to 3 centimeters on moving the body from the right lateral to the left lateral position. In cardiac fixation, there is no shift detectable either by percussion or X-ray. The systolic retractions of importance are three in number, that of the apex, Broadbent's sign, and Wenkebach's sign. Systolic retraction of the apex is often accompanied by a diffuse impulse. Broadbent's sign is a retraction of the eleventh interspace posteriorly on the left side, due to the tug of the diaphragm attachment in diaphragmatic adhesions. Wenkebach's sign is a lag of the left nipple behind the right in chest movements on inspiration. Sometimes the adhesions are so extensive as to cause a systolic retraction of a large area of the chest wall. In the electrocardiogram, there is no resultant shift of the electrical axis upon change of position noted above, due to the fixation of the heart. Constriction of the heart and great vessels results in increased venous pressure with its edema, lowered systolic pressure, and narrowed pulse pressure. Paradoxical pulse is also a common finding. Constriction of the inferior vena cava results in hepatic engorgement, with enlargement of the liver and an ascites much more severe than the rest of the edema would warrant. This disproportionate ascites, coupled with any of the other signs of constructive pericarditis, makes the diagnosis of Pick's disease. In the X-ray is noted an irregularity of outline and, rather rarely, calcification. Enlargement is rather frequently found, especially where valvular disease coexists. In the electrocardiogram low voltage of the complexes is a rather consistent finding, and inverison of all T waves is characteristic but less common. Other signs are those mentioned with effusion.

e. Prognosis.—The prognosis is good in those grades of severity not showing symptoms or signs. Frequently, these people live completely normal lives unaware of the existence of any pathology. However, those with marked involvement are usually cardiac cripples for a varying period of time, often many years prior to death. These people die either of cardiac exhaustion in middle age or at some earlier period of accident or intercurrent infection. All gradations of prognosis exist between the two extremes quoted.

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f. Treatment.—No treatment is needed except in the more serious cases. Medical treatment has little to offer except in the establishment of a cardiac regime and symptomatic relief. All of the advanced cases will be cardiac cripples and will, of necessity, be forced to live within the limits of their cardiac reserve to avoid congestive failure. Limited activity, limited diet, limited fluid intake, and the administration of diuretics are the basis of medical treatment. Relief of distress by paracentesis of the abdomen and thorax is indicated. Surgery, on the other hand, although yet in the formative stage, has something very definite to offer. Two operations are possible for the relief of constriction. In the simpler and older, known as Brauer's cardiolysis, the ribs are cut away over the area of adhesion. This relieves the heart of considerable load in that it will then tug only on soft issue rather than the more immovable bony thorax. This is successful and will prolong the life and give added comfort for a period of years only if there is no constriction of the great vessels. If constriction of the vessels or of the heart itself is present, the more extensive Delorme operation must be performed. This consists of cutting away pericardium and constricting adhesive bands, with the result that pressure is taken off the structures involved. This has been quite successful in relieving the worst features of the mediastinal syndrome in selected cases, and should be given consideration in all advanced stages of the disease.

121. Recommended texts.

Heart Disease (White)	Macmillan.
Diseases of the Heart (Lewis)	Macmillan.
Clincal Heart Disease (Levine)	Saunders.
Synopsis of Diseases of the Heart and Arteries (Hermann)	C. V. Mosby.

Section VII

THYROID HEART DISEASE

Paragraph
General ---------- 122
Thyrotoxic heart disease ---------- 123
Myxedema heart ---------- 124
Recommended texts ---------- 125

122. General.—The heart is involved in thyroid disease in only two entities, hyperthyroidism and myxedema. In hyperthyroidism the heart is affected by either an abnormal or excessive thyroid secretion. In myxedema the diminished thyroid secretion creates the untoward effect upon the heart. The two actions upon the heart

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are diametrically opposed, the increased metabolism causing havoc in one and the decreased metabolism causing the changes in the other.

123. Thyrotoxic heart disease.—*a. Etiology.*—There is but one cause for this condition, disease of the thyroid gland, which produces either an excessive or an abnormal secretion. This disease entity can occur at practically any age, although its greatest peak of incidence lies between the ages of twenty and thirty, with the decade between thirty and forty running a close second. Females far exceed males in involvement, the ratio being practically five to one.

b. Pathology.—The effects upon the heart are due to the increase in metabolism caused by the aberrant thyroid secretion. The increased metabolism results in an increased general circulation, slight peripheral vasodilatation, increased per minute blood flow, increased blood pressure, and a rapid pulse rate. The metabolism of the heart muscle itself is also increased proportionately. Both of these factors impose a tremendous increase in the load on the heart, which increased load results in dilatation, hypertrophy, and ultimately congestive failure. There are no permanent changes produced in the heart itself which are pathognomonic of hyperthyroidism; those present are entirely the end results of overwork. It should be mentioned in passing that another school of thought states that even these changes do not occur unless there is a coexisting independent cardiac condition such as hypertension, arteriosclerosis, or valvular heart disease.

c. Symptoms.—There are no symptoms typical of the cardiac condition itself. The earlier symptoms are those of hyperthyroidism, plus the effort syndrome induced by the increased metabolism: palpitation, dyspnoea, and precordial ache. The palpitation is due to the forceful beating of the heart in combination with extrasystoles or the arrhythmias. The dyspnoea is of the mild type, and not to be confused with that of venous congestion. The precordial ache is usually slight and diffuse, although in those who have a predilection to angina pectoris, that condition is apt to be precipitated with marked severity and frequency. When congestive failure sets in, the symptoms characteristic of that condition supersede the other milder ones noted above.

d. Signs.—(1) The following signs are noted in well-marked cases of thyrotoxic involvement of the heart: tachycardia, increased cardiac force, blood pressure changes, vasodilatation, arrhythmias, and murmurs. The tachycardia averages between 100 to 120 at rest or during sleep, and much more than that during exercise and excitement. The

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increased force of the heart beat is manifested by a marked thrust against the chest wall, creating a forceful slapping PMI. Frequently there is a resultant thrill over the apex and, practically always, a marked overactivity as seen by fluoroscopy. The first heart sound, especially at the apex, is markedly accentuated. Blood pressure changes consist of a moderately increased systolic pressure, slightly lowered diastolic pressure, and a resultant widened pulse pressure. The vasodilatation noted is the summation of both the increased tissue metabolism and the widened pulse pressure. In some cases the pulse pressure becomes so great that these cases are easily mistaken for aortic regurgitation. Arrhythmias of all types occur. The most important of these is auricular fibrillation. This may exist either in permanent or transient form. Paroxysms of auricular fibrillation, while not entirely pathognomonic of hyperthyroidism, should always bring that condition to mind for consideration. Fibrillation is very common, occurring in from one-third to one-fourth of all cases of thyrotoxicosis. The incidence increases with age, and is very high in cases giving a history of previous rheumatic fever. It is characterized by a very rapid ventricular response, which ventricular rate either fails to respond to digitalis administration, or does so in a very slight degree. Murmurs are a fairly common finding and are almost always systolic in time, occurring either at the apex or at the base. A fairly typical murmur is the harsh, loud, prolonged systolic pulmonary murmur due to the increased pulmonary circulation. This increased pulmonary circulation also results in a prominence of the pulmonary conus seen in the X-ray in many cases. Later in the course of the disease the above-given signs are complicated by dilatation, enlargement, congestive failure, or the arrhythmias. The electrocardiograph fails to give any findings distinctive of the condition.

(2) The dignosis of this condition in the presence of an increased basal metabolic rate, thyroid enlargement, and exophthalmos is fairly easy. However, there are many cases, called by Levine masked thyrocardiacs, in whom the diagnosis is much more difficult. These cases are chiefly those with varying degrees of congestive failure or angina, which have as their basis an undetected atypical thyrotoxicosis. Remarkable results in treatment of these cases can be made if the possibility of masked thyrotoxicosis is borne in mind and looked for in all cardiacs who are making poor or unusual responses to orthodox treatment. Levine notes that chronic cardiacs possessing an unusual alertness and quickness of movement, not ordinarily seen in the cardiac cripples, should be suspected at once of a thyrotoxic basis. Reference is made to Levine's text for more complete details on this subject.

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e. Prognosis.—Prognosis in the properly treated cases is excellent. This is one condition in which something constructive and of a permanent nature can be done for persons seemingly hopeless cardiac cripples. Complete return to activity is a common result following operation in these cases. On the other hand, the untreated cases proceed on to death, from congestive failure or fibrillation in the course of 2 or 3 years. Intercurrent infection frequently terminates the course of the disease prior to congestive failure, due to the lowered resistance.

f. Treatment.—There is but one satisfactory treatment, subtotal thyroidectomy. A successful operation gives complete functional relief in the greatest majority of cases. This condition is the one nonemergency in which operation is indicated in the presence of congestive failure or fibrillation. Likewise, this condition is one in which digitalis notably fails, whereas iodine administered preoperatively produces some startling results in congestive failure. After operation digitalis again becomes effective. In all cases, cardiac or otherwise, Lugol's solution, 4 minims (ten drops), t. i. d. for 10 days, is indicated in preparation for operation. This regime should be continued longer in the severer congestive failures if improvement continues to be manifested. It should likewise be continued for 2 weeks after operation. Congestive failure and agina are practically always relieved within 2 weeks following operation. However, fibrillation is not always broken although digitalis now becomes effective and reduces the ventricular rate to within economic limits. Frequently, in the transient type of fibrillation, an attack will set in shortly after operation which will prove unusually troublesome. These usually respond eventually to digitalis.

124. Myxedema heart.—The heart involvement in myxedema is due to the reduced thyroid secretion and the resultant lowered metabolism. It is seen more frequently in middle age or later. Like the hyperthyroidism, there is no definite pathology to be seen in the heart. Also, like the hyperthyroid state, there are no symptoms characteristic of the cardiac involvement, merely those of the underlying disease being prominent. Signs are chiefly those of a markedly enlarged heart both to percusssion and X-ray, the condition in the more severe cases being often mistaken for pericardial effusion. A very noticeably sluggish action of the heart is another sign. The electrocardiogram shows a low amplitude in the QRS group and a flattening, inversion, or isotonicity of the T wave in all leads. Otherwise, the signs are those of myxedema, with its lowered BMR and sluggish activity. The prognosis is good with treatment. Thyroid

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extract administered in proper amounts produces in a short time a very startling reduction in heart size, a return to fairly normal cardiac activity, and a restoration of amplitude in the electrocardiogram. Sudden death is a frequent accompaniment in the untreated cases.

125. Recommended texts.

Clinical Heart Disease (Levine)---------- Saunders.
Heart Disease (White)---------- Macmillan.
Diseases of the Heart (Lewis)---------- Macmillan.

Section VIII

NEUROCIRCULATORY ASTHENIA

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General ---------- 126
Etiology ---------- 127
Pathology ---------- 128
Symptoms ---------- 129
Signs ---------- 130
Prognosis ---------- 131
Treatment ---------- 132
Army Regulations ---------- 133
Recommended texts ---------- 134

126. General.—Neurocirculatory asthenia is an instability of the nervous system manifested by signs and symptoms referable to the cardiovascular system. It is to be emphasized that it is a neurasthenic syndrome and not an organic disease of the heart. Its importance lies in the frequency with which it is confused with heart disease. This confusion arises because of the similarity of its signs and symptoms to those of certain cardiac conditions. Neurocirculatory asthenia is of importance to flight surgeons because it is a distinct manifestation of basic instability and persons manifesting such instability are notoriously dangerous to themselves and others when placed in flying capacities. Therefore, its early recognition in flying cadet applicants is very essential.

127. Etiology.—The exact cause of neurocirculatory asthenia is unknown. Essentially it is one of the three manifestations of neurasthenia which are the gastro-intestinal type, the cerebral type, and the cardiovascular type. Therefore, the numerous etiological factors of neurasthenia apply in this condition. Young adults between the ages of twenty and thirty-five are those most affected. Heredity has a very definite bearing, and in taking histories prior to the examination of flying, any family history at all suggestive of nervous instability or subnormal nervous make-up should put the examiner instantly on the alert.

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128. Pathology.—There is no pathology found in the heart which can be considered pathognomonic of this condition.

129. Symptoms.—There are many symptoms to be noted, the chief of which are palpitation, respiratory discomfort, precordial aches and pains, exhaustion, faintness and fainting, dizziness, tremor, sweating, nervousness, and a feeling of insecurity and inferiority. White states that palpitation, respiratory discomfort, precordial discomfort, and exhaustion are the four most important of the symptoms, and that they will be seen in from 78 to 73 percent of cases, respectively. The palpitation, often bitterly complained of, is due to the rapid action of the heart and frequent extrasystoles. The dyspnoea is entirely subjective and is more a sense of suffocation than a real shortness of breath. Its distinguishing features are respiratory discomfort without respiratory effort, plus frequent sighing. Sighing is notably absent in organic heart disease. The precordial discomfort is essentially a dull ache in the left chest, often lasting for hours or days. Very rarely does this pain reach the severity of angina with which it is often confused by the patient. Exhaustion is especially to be noted in that these individuals tire very easily, are physically inefficient, and lack the coordination and stamina of the ordinary person. For this reason alone, they are not amenable to flying training. The other symptoms do not need remark, except that dizziness is often noted on quick changes of position and complained about especially.

130. Signs.—*a.* The signs likewise are numerous, and are not all present in any individual case. They are tachycardia, tremor, excessive perspiration, blue cold congested extremities, unstable blood pressure, functional systolic murmurs, increased force of the heart action, diffuse PMI, sinus arrhythmia, flabby musculature, less than average strength, easy fatigue, lowered vital capacity, and a low Schneider index. An "anxious expression" has also been described by some writers as a fairly constant finding. The pulse rate is frequently 90 to 120 at rest. Exercise often increases it markedly and the pulse then returns to resting normal very slowly. The opposite effect sometimes occurs after exercise. In some cases the pulse rate returns to a normal range in a perfectly normal manner following rest after exercise, and then very slowly increases to its previous rapid resting rate. The most notable point in diagnosis of this condition is that the pulse and blood pressure are normal during sleep. The coarse "nervous" tremor of extended fingers, eyelids, tongue, lips, and often entire muscle groups is sometimes very

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marked. The profuse sweating is a very prominent feature and these individuals are often seen with perspiration dropping from axilla and forehead while at rest. The sluggish peripheral circulation is often very noticeable and the finding of cold, clammy, blue, mottled, sweaty hands and feet will almost make the diagnosis alone. Infrequently, there is a tachypnoea. The X-ray and electrocardiograph offer no help at all in this condition, and all other signs of organic heart disease are notably absent.

b. It should be mentioned in passing that these symptoms and signs resemble those of the effort syndrome in considerable degree. Effort syndrome is that response to severe exertion occasioned in normal individuals, that is, tachycardia, labored breathing, sweating, and fatigue. In neurocirculatory asthenia the effort syndrome is invoked by much less effort and exertion than in normal individuals, or by no exertion whatsoever. The mild tension and excitement of the examination for flying usually bring out the syndrome in the former type to the point where a diagnosis can easily be made.

c. Lesser degrees of neurocirculatory asthenia, involving especially an unstable blood pressure, unstable pulse, coarse tremor, and a low Schneider index, are classified by the author as "vasomotor instability." The cold, blue, congested extremities and muscular flabbiness are frequently absent and the individual has the appearance of being normal except for the points mentioned above. In these cases, the systolic blood pressure has been observed to vary in seven or eight consecutive readings over a range of 60 points, no two ever corresponding. The pulse rate has likewise been seen on innumerable occasions to vary over a range of 50 points during the different portions of the examination in which no exercise is involved. Less often, other signs will be noted which will aid in making a diagnosis, especially the profuse sweating at rest, or a marked subjective tenseness. An unstable pulse and blood pressure, plus tremor, certainly justify the diagnosis of vasomotor instability when the symptoms and findings are not severe enough to warrant that of neurocirculatory asthenia.

131. Prognosis.—The prognosis as to length of life is excellent. There are no fatal aspects to this condition. However, the trouble is a very real one to the individual concerned, and often occasions considerable suffering and apprehension. All of this results in a rather ineffectual, neurotic type of existence.

132. Treatment.—There is no treatment indicated, except the pyschotherapy employed in the psychoneuroses. Medication and restriction of activity are definitely contraindicated, in that they

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merely confirm the patient's already too firm conviction that there is something organic as the basis of his troubles. Treatment should be confined to such measures as reassurance, aid in solving situational difficulties, exercise, and other general measures aimed at regaining self-confidence and stability. However, except in rare instances, very little of lasting good will be accomplished even by these measures, and the individual will go on throughout his life with his unstable nervous system ruining his physical and mental comfort. As soldiers, these people are of little or no value to the Army, and it is very frequently necessary to separate them from the service by discharge for disability. As prospects for flying training, or for duty as military aviators, they are hopeless, and it is every examiner's duty to spare the Army the expense of even beginning their training. The occupation and duties of a military pilot require a remarkable degree of stability and stamina, both mental and physical, which even those persons exhibiting the lesser degrees of circulatory neurasthenia, which we have labeled vasomotor instability, do not possess.

133. Army Regulations.—Paragraphs 31*f*(3), AR 40–110 states "On the original examination a definite diagnosis of neurocirculatory asthenia disqualifies." In paragraph 31*f*(1), AR 40–110, the following statement is made: "In the case of applicants for flying training, a persistent systolic blood pressure of 135 millimeters or more, or a persistent diastolic blood pressure of 90 millimeters or more, or an unstable blood pressure disqualifies."

134. Recommended texts.

Heart Disease (White)---------- Macmillan.
Clinical Heart Disease (Levine)---------- Saunders.

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CHAPTER 4

DISEASES SEEN IN OLDER PILOTS

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SECTION I

CARDIOVASCULAR SYPHILIS

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135. General.—Cardiovascular syphilis is of importance because of the physical disability it creates in personnel who have had from fifteen to twenty or more years' service, and who, because of the condition, become dangerous to themselves and others while on flying status. This is especially true in those cases in which the initial or secondary lesions were undetected or unsuspected and the disease, because of its insidious advance, is quietly accomplishing irreparable and unrecognized damage within the victim's aorta. It is also to be considered that because of the adverse line-of-duty findings attached to the acute condition, certain persons will have undergone sub rosa, and probably insufficient treatment, and may still be taking steps to conceal their condition. Therefore, it behooves the examiner to be especially alert and suspect all persons in their own interest. Routine blood Wassermann or Kahn tests on all flying personnel at least as frequently as every 3 years are recommended in order to diagnose the disease earlier and thus be enabled to prevent some of the cardiovascular damage before it becomes incurable.

136. Etiology.—*a.* Cardiovascular syphilis is a fairly common condition, being one of the four principal causes of cardiovascular disease. It is especially frequent in the south among the negroes in whom from one-fourth to one-third of all heart disease has its origin lues. The condition is also common in the white race and has a

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wide distribution throughout all ranks of society. In general, however, the poorer classes with their poorer environmental conditions and consequent lower moral standards, suffer a higher incidence of the disease.

b. There is only one cause of the disease, the treponema pallidum. The cardiovascular condition is seen principally in the tertiary stage of the acquired type, and very rarely in congenital lues. Symptoms and signs usually appear in from fifteen to twenty years after the initial luetic lesion. Thus the age groups forty to sixty are those in which cardiovascular syphilis is most commonly seen. Except in negroes, the condition is rather uncommon before the age of forty or after sixty. Males have been found to have this luetic complication much oftener than females, the ration being approximately five to one. It should always be remembered that even though Army officers are as a group of much higher intelligence than the average, the disease is no respector of persons, and that because of the reasons stated in paragraph 135, steps should never be omitted which might bring the condition to light.

137. Pathology.—*a.* The spirochetum pallidum seems to have an affinity for the cardiovascular system in a certain percentage of cases, just as it seems to have an affinity for the central nervous system in other cases. The best opinion today holds that this organism invades the cardiovascular system shortly after the initial lesion, and is present throughout the entire period of latency prior to the manifestation of symptoms and signs. The spirochete invades both the larger arteries, especially the aorta and the myocardium. The basic pathology of the arterial invasion is inflammation and destruction of the media, with its attendant consequences. The basic pathology of the myocardial invasion is of two types, a diffuse inflammatory involvement and the production of gummata. Both are rather rare types of lesions and will be missed prior to autopsy more frequently than not, unless there is some resultant involvement of the conducting system to produce the various types and grades of heart block. The diffuse inflammation has been reported as being the cause of sudden death in several instances where cardiovascular disease was not previously suspected.

b. The inflammation and destruction seen in the arterial type of syphilis center about the root of the aorta. Here the earliest lesion is an obliterating arteritis with a perivascular round cell infiltration of the vasa vasorum. The result is an obliteration of these nutrient vessels with destruction of the tissue in the media normally supplied by them, especially the elastic tissue. This loss of medial

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substance leads to fibrosis and puckering of the overlying intima and results in the characteristic intimal wrinkling of syphilis. This wrinkling is a longitudinal type of striation in the intima which alone is almost diagnostic of the disease in the absence of other findings at the autopsy table. These striae, which are whitish or grayish plaques, become progressively less in number as one proceeds distally along the arterial tree from the aortic ring. In the ascending aorta they may be so numerous as to appear confluent. The greatest functional result of this destructive aortic process is the loss of elasticity of the aortic wall. This loss of elastic tissue causes stretching and dilatation of that vessel. Upon this loss of elasticity and stretching depend two additional later manifestations of cardiovascular syphilis, aneurysmal dilatation and insufficiency of the aortic valve. (Syphilis is the direct cause of approximately 90 percent of all aneurysms of the aorta.) A fourth, and rather rare result of the aortic inflammation, is the involvement of the mouths of arterial branches arising from the aorta in an obliterative fibrous process which often ends in stenosis. The aortitis in itself is of little significance except as a forerunner of the more serious consequences outlined.

c. The aneurysmal dilatation is of two types, saccular and diffuse or fusiform. Both types arise most frequently in the ascending aorta but may also be found in the other large vessels. Aneurysms place very little additional burden upon the heart, and often are not detected prior to autopsy. Frequently, the saccular type is filled with a blood clot. The chief manifestation of either type is that caused by the pressure on neighboring structures such as trachea, esophagus, mediastinal nerves, or bony erosion. The dynamic force of an aneurysm always destroys, displaces, distorts, erodes, or compresses any structures which happen to lie in the direction of its growth and extension. Rupture of an aneurysm is a not uncommon form of death in this complication of lues.

d. Insufficiency of the aortic valve is much more common. The process is due to two factors, extension of the inflammatory process onto the valve cusps and dilatation of the valve ring. The linear plaques described under aortitis extend down the aorta to the valve attachments and tend to separate the cusps at their bases and to thicken the commissural edges. This leads to improper closure of the cusps and aortic insufficiency. The stretching of the valve ring adds insult to injury in this case, further widening the space between the cusp margins. The end results of aortic insufficiency, enlargement and congestive failure, have been described elsewhere.

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e. The obliteration of the vessel mouths most frequently and seriously involves the coronaries. Here the coronary ostii are involved in the extension of the inflammatory luetic process down the aorta into the sinuses of Valsalva. The wrinkling and plaque formation involves the mouths alone and rarely if ever extends down into the coronary vessels themselves. The end result is a decreased coronary circulation with subsequent inefficiency of the heart, angina pectoris, and at times sudden death from mechanical occlusion of the mouths themselves. The same process is noted much more infrequently in the arteries arising in the transverse and descending aorta in the same diminishing frequency that the basic process extends away from the aortic root.

138. Symptoms.—The consideration of both symptoms and signs of cardiovascular syphilis will be undertaken on the basis of the four main divisions of the disease described above:

a. Aortitis is most often symptomless. Sometimes there is a dull ache under the upper sternum, which varies somewhat in severity but is more or less constantly present and has very little relationship to exertion. This remarkable lack of symptoms is due to the very small additional burden which uncomplicated aortitis places upon the heart.

b. Aneurysm of the aorta presents many more symptoms but, like aortitis, places very little additional work upon the heart. There are usually no symptoms, other than the dull ache described above, until the aneurysm becomes large enough to exert significant pressure upon surrounding structures. In fact, all symptoms of significance in this condition arise as a result of pressure. Pain and breathlessness are the two of most importance. Pain is often very severe and due to either erosion of bony structures such as vertebrae, ribs, sternum, and clavicles, or to pressure on neighboring nerve structures. The breathlessness is due to compression of the lungs, trachea, or bronchii when that compression reaches a degree sufficient to constrict materially the volume of air in respiration. A dry, brassy type of cough is to be seen when pressure on air passages is sufficient to cause irritation or when certain nerves are impinged upon. Hoarseness and later aphonia arise due to pressure upon laryngeal nerves. Dysphagia is the result of pressure upon the esophagus and in some cases becomes severe enough to produce semistarvation. Hemoptysis occurs when ulceration or erosion arises in trachea, bronchii, or lungs, or when there is a slow pinpoint leak from the aneurysm itself into one of those structures. Pressure on blood vessels gives symptoms peculiar to the area served by the vessel compressed.

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c. Aortic regurgitation and the symptoms arising from its results, heart strain, enlargement, and failure, have already been discussed.

d. Blockage and constriction of the mouths of the coronary vessels give rise to angina pectoris of varying degrees. This is due to the insufficient coronary circulation and the resulting myocardial ischemia. Very rarely cardiac infarction occurs as the result of a sudden complete blockage of one of the mouths. Sudden death results in rare instances from this same phenomenon. When aortic branches other than the coronary arteries are occluded or obstructed, the symptoms arising are those peculiar to the area supplied.

e. In general, cardiovascular syphilis is a silent type of condition and often the first indication of its existence may be angina, paroxysmal nocturnal dyspnoea, or a Cheyne-Stokes type of respiration. Congestive failure or even sudden death may be the first indication that the heart or aorta is infected with syphilis. In syphilitic affections of the myocardium, symptoms are rarely present, and those which do exist are due to the varying degrees of heart block which arise.

139. Signs.—*a.* Cardiovascular syphilis is devoid of signs during the period of invasion and often there may be no signs whatsoever even in the far advanced stages. It is not uncommon for the pathologist to make a diagnosis of cardiovascular lues in cases where its existence was not at all suspected. When signs do appear, the case is in the advanced stage, the damage done, and little hope of constructive treatment remains.

b. Aortitis is especially notable for the absence of signs denoting its existence. The heart is usually of normal size inasmuch as the simple inflammation of the aorta and the early mild dilatation impose no additional burden. There are two signs, both more or less inconstant, which are indicative of the trouble, a harsh aortic systolic murmur and a ringing, accentuated, tympanitic second aortic sound, which is described as having a "metallic" quality. Both of these signs are found in other conditions but their occurrence indicates the possibility of aortitis.

c. Aortic aneurysm likewise may be entirely silent. Like aortitis, an aneurysm imposes very little additional burden upon the heart unless the aortic valve is also involved, and there is little or no cardiac enlargement. When signs do occur they are entirely characteristic of the condition. The four principal signs are abnormal dullness at the base of the heart, localized pulsation over the upper chest, evidences of compression of the interthoracic organs, and an X-ray finding of enlargement or sacculation of the aorta. The finding

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of abnormal areas of dullness at the base of the heart needs no comment. The localized pulsation is seen most commonly in the second right interspace at the sternal border. Less commonly it may be seen anywhere in the anterior chest, neck, or sternum, or even in the posterior portions of the chest. The pulsation is practically never seen in the chest below the level of the manubrium sternum. Pulsations are systolic in time and often more visible than palpable. In this connection the examiner is often surprised at the apparent lack of force in a pulsation which appears to be unusually vigorous. Inasmuch as the growing dynamic aneurysm often erodes through structures in its path, especially bone, the pulsations are usually tender and painful and the skin over them is inflamed. Pulsations are frequently preceded by localized bulgings, such bulgings being the earlier manifestation of the appearance of the aneurysm at the surface.

d. Besides eroding through the more resistant structures, aneurysms displace and compress the softer tissues. This results in a wide variety of signs, depending upon the structure displaced or compressed. Most of the compression phenomena occur when the aneurysm is located in the transverse portion of the aorta. If it is remembered that the aorta arches over the left bronchus, most of the following phenomena will be easily explained. Tracheal tug, due to systolic displacement of the left bronchus downward with resultant pull on the trachea, is very prominent. Breathlessness and stridor are frequently the result when the trachea itself is compressed. Compression of the left bronchus gives signs of deficient aeration of the left base, absent or diminished breath sounds, resonance, and later infection of the area. Compression of the lung is seen in the right apex mostly and manifested by the signs of atelectasis. Compression of the esophagus is very well brought out by X-ray procedures and barium paste. All of these organs may be involved in erosion and produce varying degrees of hemoptysis and hematemesis. Pressure on nerve structures is very frequent. When the recurrent laryngeals are involved there is hoarseness and later aphonia. When the left sympathetic is involved, the left pupil contracts, the left eyeball recedes, and the palpebral fissure narrows. This inequality of the pupils is one of the most characteristic of the pressure signs. Pressure on blood vessels gives the characteristic signs of insufficient circulation in the area supplied. Pressure on the superior vena cava gives a fairly typical upper mediastinal syndrome, but this condition in syphilis is rather rare. X-ray finding of diffuse dilatation of any portion of the aorta or sacculation will make the diagnosis in the presence of a positive Wassermann. In fact, the finding of sacculation

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is almost absolute evidence of the syphilitic type of aneurysm. Fluoroscopy to detect pulsation of any dilatation or bulging is especially informative. Additional signs which should be mentioned are inequality of the radial pulses and inequality of blood pressure in the two arms. These are due to aneurysm arising at some point between the origins of the two subclavian arteries or in one of the arteries itself. The harsh aortic systolic murmur noted in aortitis is sometimes present, as well as the ringing metallic second aortic sound. When present, the murmur in the aneurysm itself sometimes assumes a to and fro characteristic.

e. The signs arising in aortic insufficiency have been discussed elsewhere. Briefly, they are marked cardiac enlargement, loud aortic diastolic murmur, absent aortic second sound, Austin Flint murmur, Corrigan pulse, and an extremely wide pulse pressure.

f. Constriction of the mouth of the vessels gives no signs other than the angina. Complete occlusion is rather rare and usually of slow onset and consequently gives rather misleading signs of the condition. In any angina, a Wassermann is indicated before making a final diagnosis.

g. Blood serology is an essential part of the group of signs. The Wassermann is positive in approximately 85 percent of cases and the finding of any signs or symptoms even suggestive of aortic pathology, plus the presence of positive serology, emperically make the diagnosis. While it is possible for lesions due to other causes to exist at the base of the heart in the presence of positive serology, the chances are overwhelming that there is an interrelationship between the two, and cardiovascular lues cannot be ruled out without very convincing evidence to the contrary. The electrocardiograph is of no help whatsoever in this condition until the muscle becomes involved, either primarily or as an end result of the other lesions.

140. Prognosis.—Cardiovascular lues is a disease of unusually slow and insidious onset, requiring from fifteen to twenty-five years before symptoms are manifest. When signs and symptoms do develop, the disease is already in an advanced stage and the prognosis is therefore poor. In untreated cases, a diagnosis of cardiovascular syphilis means that the patient has from a few months to 2 or 3 years to live, rarely longer. Treatment, especially the proper type of antiluetic therapy, has improved this prognosis somewhat, and tends to prolong life. However, too vigorous treatment will hasten death appreciably in many cases. Death occurs suddenly from congestive failure or from intercurrent infection. All of the individuals will be cardiac cripples of various degrees during the remainder of their lives after symptoms and signs are definite enough to make an absolute diagnosis.

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141. Treatment.—*a.* Treatment of the cardiovascular condition is that of any cardiac disability plus specific therapy. A regime including rest, avoidance of excesses, and the administration of symptomatic relief is indicated. Nitrites, digitalis, and diuretics are to be used freely when indicated. Specific antiluetic therapy is to be given in selected cases when there is no kidney lesion, no liver disease, and no congestive failure present. In the presence of such conditions, specific therapy merely hastens disability and death. Congestive failure can often be relieved, and when compensation is restored and the other two contraindications are absent, heavy metals and arsenicals can be begun. In passing, it should be noted that the best diuretics in existence, the mercurials, salyrgan, mercupurin, and allied drugs, are also very effective as antiluetics, and their administration in congestive failure will often accomplish a double purpose.

b. The antiluetic therapy should begin with a course of heavy metals, preferably bismuth in 0.1-gram doses per week for 12 weeks. This should be followed by a course of arsenicals in small doses, preferably neosalvarsan in 0.1-gram to 0.2-gram doses per week for a period of 12 weeks. These two courses should be alternated without intermission for the remainder of the patient's life, as long as toxic symptoms do not appear. Administration should stop immediately that any of the toxic symptoms do develop. It is to be remembered that this dosage is merely one of a multitude of plans of antiluetic cardiac therapy, and that there are many others offered which are just as successful in the hands of their users. Some prefer larger doses, or increasing doses, or the old "mixed treatment," or potassium iodide. All are useful if properly handled. If antiluetic medication is given with caution, taking especial care to avoid the pitfalls noted above, a prolongation of life up to several years and a large degree of relief from symptoms can reasonably be expected.

c. In aneurysm, surgery is sometimes indicated. Wiring of saccular aneurysms is done with the hope of establishing a firm clot for the purpose of obliteration of the cavity. Sometimes electrolysis is combined with wiring, many authorities holding that electrolysis hastens clot formation and produces a firmer type. It is also possible in some cases to support the aneurysm with a sheet of dense fibrous tissue, such as a piece of fascia lata. This, however, is a very difficult technical procedure and of somewhat doubtful value. In the smaller aneurysms of peripheral vessels, ligation of the vessel by stages can be done with the idea of establishing a collateral circulation for that extremity.

d. The value of early antiluetic therapy as a prophylactic measure is rather commonly underestimated. Timely and adequate antisyphi-

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litic treatment begun shortly after the development of the primary lesion is of greatest importance as a preventive of cardiovascular syphilis in later years. The general incidence of cardiovascular syphilis has remarkably decreased since better treatment has been instituted in the past 20 years. Statistics recently published also show that cardiovascular involvement occurs much less frequently in treated than untreated cases. Therefore, the importance of comprehensive and complete specific therapy at the hands of a qualified physician should be emphasized, if only as a method of preventing future cardiovascular damage.

142. Recommended texts.

Heart Disease (White)---------- Macmillan.
Synopsis of Disease of the Heart and Arteries (Hermann)---------- C. V. Mosby.
Clinical Heart Disease (Levine)---------- Saunders.
Diseases of the Heart (Lewis)---------- Macmillan.

Section II

ARTERIOSCLEROSIS

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General---------- 143
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Symptoms---------- 146
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Dissecting aneurysm---------- 150
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143. General.—Arteriosclerosis is that degenerative process which occurs in the arteries of the body as a part of the advance of senescence. All of the arteries from the largest to the very smallest are subject to its involvement. It is very common, being one of the four major causes of heart disease. Its importance lies in the fact that it is a disease of older persons, and those officers or flyers subject to its ravages suffer a definitely reduced cardiovascular efficiency. Its local effects on specific organs such as brain, heart (coronary circulation), and kidneys make it one of the greatest causes of disability.

144. Etiology.—*a.* The cause of arteriosclerosis is unknown. Many have been listed at various times in the past, and the very multiplicity of the possibilities adequately confirms the fact that the real cause is as yet unknown. Two factors, heredity and the wear and tear of existence, are of most importance. Susceptibility to arteriosclerosis seems to be inherited, and it has often been shown that the

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tendency to either early onset or especial severity seems to run in families. In other words, the quality of the arterial wall of an individual depends to a large degree upon the quality of the arterial walls of his ancestors. The factor, wear and tear of existence, is complicated by many subfactors. The mere living of even an uncomplicated life ultimately produces degenerative arterial changes of some degree. Some authorities hold that the natural pressure maintained in the arterial tree will cause sclerosis. The stresses and tensions of modern civilization with its more strenuous living, hard work, and overindulgence seem to add to the burden of the arteries. Overeating and excessive use of tobacco, alcohol, and coffee are likewise thought to have a deleterious effect. Foci of infection, metabolic disturbances, especially diabetes, and the infectious diseases also seem to have a bearing. Hypertension is definitely known to cause thickening of the media of the peripheral arteries. On the other hand, arteriosclerosis is not a cause of hypertension, and although the two are more often than not associated, arteriosclerosis frequently exists in the absence of hypertension. None of these factors alone is of prime importance and in any given case they usually exist in groups, making it very difficult to assign any single one as cause. The most acceptable individual theory yet brought forward is that the condition is due to a faulty cholesterol metabolism.

b. The process is an accompaniment of old age, being seen principally after the age of forty-five. The frequency of incidence increases as the age groups advance. However, it is not limited to old age and Lewis maintains that after the age of thirty the arteries are no longer strictly normal. It is relatively common to find atheromatous changes in an aorta at autopsy in younger individuals who have died from other causes.

145. Pathology.—*a.* Arteriosclerosis is a degenerative lesion, and all pathology seen is basically degeneration and subsequent repair. This process occurs in two types, the atheromatous, seen principally in the larger vessels and the hyperplastic, seen principally in middle sized and smaller arteries. The condition can be either generalized or localized. When localized, the arteries principally affected are those of the brain, heart, kidneys, pancreas, or extremities.

(1) In the atheromatous type, the earliest lesion is a deposit of minute amounts of fat in and beneath the cells of the intima. These deposits increase in size, become yellowish subendothelial patches, and create small raised areas in the intima. The reaction to these degenerative patches is a surrouding fibrosis which, as it develops and becomes extensive, results in a hardening and inelasticity of the areas involved. In the aged, deposits of calcium very frequently are found

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in these areas of fibrosis. As the condition progresses, larger areas of the arterial walls become involved. In the aorta the entire circumference may become a thickened, irregular, rigid, inelastic, fibrous mass. The descending aorta is the center of involvement, the abdominal aorta being next most extensively involved. The ascending aorta is frequently entirely clear of all atheromatous lesions even when the descending portion is extensively affected. This is just the opposite to the aortic findings in syphilis, in which the center of the lesion is at the root of the aorta and the major involvement in the ascending portion. One other point of differentiation between the two is that the atheromatous plaques tend to be irregular in outline and disposed somewhat transversely, whereas the intimal markings of syphilis are linear and disposed longitudinally. Smaller vessels are also involved by atheromata, and the smaller the vessel, the more the atheromata encroach upon the lumen and diminish the flow of blood to the parts supplied. In the terminal stages of the atheromatous process, the calcerous plaques sometimes fracture, or the fat deposits, which are mostly cholesterol, ulcerate. Over these lines of fracture or ulcerations, thrombus formation takes place. In the larger vessels these thrombi tend to break off and become emboli, creating embolic phenomena at the points of lodgment. In the smaller vessels, thrombus formation means occlusion, either complete or partial, with resultant signs and symptoms of diminished blood supply in the areas distal to the closure.

(2) In the hyperplastic type of sclerosis, the media of the middle sized and smaller arteries become gradually thickened by fibrosis. Later in the course of the disease calcification occurs. This fibrosis and calcification reduces the size of the lumen of the vessels and diminishes the elasticity of the arterial wall. The result is a gradually lessening flow of blood to the areas supplied. Loss of elasticity further produces lengthening and tortuosity of the vessel, the end result of which is a stiff, lifeless tube through which the blood flows rather than is propelled. (Normal arteries are pulsating vessels whose elastic rebound to systolic stretching further aids in the propulsion of blood.) The results of this smaller vessel type of sclerosis are especially severe when seen in the brain, kidneys, and extremities. Frequently complete occlusion occurs, but often it is not acute in that the process may be so insidious that there is adequate time for a collateral circulation to develop.

b. There is one other special type of sclerosis which has its end result in an aortic stenosis. The aortic valve is the only one affected by this process. Here subendocardial fibrosis first occurs at the bases of the aortic cusps. The process extends along the valve mar-

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gins and later subendocardially in the substance of the leaflet itself. The condition progresses to complete calcification of one or more of the valve cusps with resulting deformity and obstruction to blood flow. It is one of the causes of an aortic systolic murmur in aged arteriosclerotics, and where that murmur exists, a pure arteriosclerotic aortic stenosis should always be considered.

c. The heart itself is not otherwise involved in arteriosclerosis and will show no signs or symptoms unless the coronary vessels are fairly extensively involved, or there is a coexisting hypertension of considerable degree. In either of these cases, the cardiac manifestations are those resulting from the basic condition.

d. It should be noted that there are many more diseases of the vessels than have been discussed. Lack of space or lack of application to the subject prevents their consideration. For the student's reference, a partial list is furnished: Arteriovenous aneurysm, infectious endarteritis, periarteritis nodosa, thrombo-angiitis, obliterans, Raynaud's disease, erythromelalgia, scleroderma, and Nothnagel's syndrome.

146. Symptoms.—*a.* The onset of this condition is so insidious that symptoms are usually absent until the disease has progressed to the point of interference with circulation. All symptoms, therefore, are dependent upon the disturbances of function that result from the cutting down of blood supply to the various structures. This diminution of blood flow may be very gradual with adequate time for the development of a more or less efficient collateral circulation, or it may be abrupt as in the formation of thrombi over a fractured plaque or the lodgment of an embolus.

b. Generalized arteriosclerosis is usually manifested by senility with its changes in physical and mental efficiency. The mental symptoms are usually quite prominent in that generalized arteriosclerosis usually has as an accompaniment a fair degree of cerebral involvement.

c. Localized arteriosclerosis is manifested chiefly in the brain, heart, kidneys, pancreas, and extremities. In the cerebral involvement are all of the symptoms of cerebral arteriosclerosis: irritability, mental fatigue, inability to concentrate, amnesia, especially for recent events, depression, and emotional instability. Later in the course of the disease, thrombosis may occur, or, if there is coincident hypertension, a hemorrhage. The train of mental and neurological symptoms which follow these events is too well known to reiterate here. In the heart, the findings are those of coronary insufficiency and occlusion, both of which are discussed in some detail in paragraphs 152 to 154. In the kidneys, symptoms are usually absent until the

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onset of a rather marked renal impairment or a renal insufficiency. The chief symptom of kidney involvement of the arteriosclerotic type is that of nocturia. This is often very bitterly complained of in that it further disturbs an already fitful rest. In the pancreas, arteriosclerosis of the supplying vessels gives a true diabetes, usually mild, and fairly easily controlled. In the extremities, the chief complaints are those of pain, cramps, and numbness. Pain is of the intermittent claudication type and occurs especially in the lower extremities during attempted exercise. Here the patient is able to walk for only a measured distance without rest. The anoxemia of the muscles occasioned by diminished blood supply and increased muscular demand causes excruciating pain until rest allows the blood supply to replenish the needed oxygen. Cramps at rest or during sleep are fairly common and thought to be due to some mechanical interference with blood supply or to an unexplained angiospasm. Numbness, tingling, and burning sensations are frequently present, also due to inadequate blood supply. When thrombosis or embolism occurs, the sudden cessation of blood supply causes gangrene and its accompanying train of symptoms.

d. Arteriosclerosis of the aorta, like the aortitis of syphilis, is more frequently than not symptomless. When symptoms do occur they are those of substernal oppression or pain, breathlessness on exertion, and more rarely yet, paroxysmal dyspnoea. Here, as in syphilis, aortic involvement places very little additional burden upon the heart, and the symptoms of congestive failure rarely appear unless there is some other complicating entity such as valvular disease or hypertension.

147. Signs.—*a.* Signs of arteriosclerosis appear much earlier than symptoms and the diagnosis will be made at routine examination more often than not. However, as with symptoms, by the time that signs are well developed, the disease is far along in its course. The signs of this condition will be discussed in the localized groups of their occurrence, inasmuch as a diagnosis of generalized arteriosclerosis is usually a summation of the local signs.

b. Arteriosclerotic aortitis will be recognized chiefly by auscultation and X-ray. Percussion is of no value inasmuch as the aorta is located fairly deep in the chest and is but slightly dilated in this condition. On auscultation one hears a harsh systolic murmur at the aortic area unaccompanied by a thrill. This is well transmitted into the vessels of the neck and arms. It varies widely in its characteristics, but is usually loud, harsh, rough, and prolonged. The aortic second sound has the accentuated, metallic ring which is character-

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istic also of syphilitic aortitis. The X-ray shows dilatation, elongation, and tortuosity, very characteristically described as an "unwinding" of the aorta. The aortic knob is very prominent, as is the shadow of the ascending portion. Plaques will often be visible.

c. Arteriosclerotic aneurysms of the aorta are second in importance only to the luetic type, and comprise the greater portion of the remaining 10 percent of aneurysms of that vessel. (It will be remembered that syphilis causes approximately 90 percent.) The arteriosclerotic type is mainly a diffuse dilatation, whereas the luetic type is principally saccular. The diffuse type is usually silent and like simple aortitis produces little or no additional burden on the heart. It will be detected chiefly by X-ray. The differentiation by X-ray between simple dilatation and aneurysm is merely one of degree. Fluoroscopy frequently shows a marked aortic pulsation. The systolic aortic murmur and accentuated second sound are also present. Another type of aneurysm occasionally seen in arteriosclerosis is the dissecting type, which is discussed briefly in paragraph 150.

d. Sclerosis of the aortic valve gives the signs of aortic stenosis; a systolic murmur, a systolic thrill, plateau pulse, diminished pulse pressure, and cardiac enlargement.

e. In the earlier stages arteriosclerosis of the arteries of the extremities is manifested by tortuosity and thickening. As the vessels lose their elasticity they lengthen and become tortuous, such tortuosity being best seen in brachial and radial arteries. Pulsations of these arteries may be seen over superficial areas of wrist and elbow and later along the whole course of the arteries in the arm. This phenomenon can be best accentuated by flexing the forearm upon the arm, causing the vessels to stand out. Later the vessels become very prominent subcutaneously and can easily be seen in their entire course under the skin. Thickening of the vessels can be palpated in the earlier stages. If the brachial artery is occluded and the radial palpated, the normal arterial wall cannot be felt. Any degree of infiltration or thickening of the artery will enable that vessel wall to be palpated and rolled under the finger. Later, as these vessels become thicker and deposits of calcium occur, they can be felt as "beaded" or even "pipestem." Any radial or brachial artery that can be palpated when blood is excluded is to be considered sclerotic. In the advanced stages of peripheral arteriosclerosis, when the blood supply is either gradually or suddenly cut-off, the signs of insufficient blood flow are noted. Paleness and coldness of the extremity plus absent pulses and trophic changes are the signs of this condition. Gangrene is the result of occlusion. X-ray of the tibials, radials, or brachials will often show calcification of these vessels.

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f. The retinal arteries are available for direct visual examination and here is seen the actual sclerotic process taking place. Tortuosity, crossing phenomena, beading, and silver streak findings are indicative of the condition. Later in the course of the disease, hemorrhages, exudates, and other evidences of retinal degeneration are seen. The extent of retinal arteriosclerosis is rather commonly taken as an index to the extent of cerebral involvement.

g. The signs of cerebral and visceral involvement are very numerous and all characteristic of the affected organ. In the cerebral involvement the variety and scope of the signs are astounding. Irritability, insomnia, depression, and memory defects may be the first noted. Recurring attacks of weakness, unconsciousness, or aphasia are later noted. In thrombosis or rupture of one of the cerebral vessels, monoplegia or hemiplegia may occur. Epileptiform and stuperous states are less common. Sometimes a progressive type of lesion due to a very gradual but advancing arterial diminution leads to cerebral softening with its irritability, confusion, and gradual deterioration into more or less complete dementia. The signs in sclerosis of the coronary arteries are discussed in detail in paragraphs 152 to 154. In renal sclerosis the urine will show albumin and casts in the earlier stages. Later, the signs of renal impairment and later yet those of renal insufficiency appear, that is, inability of the kidneys to concentrate, polyuria, nitrogen retention, and finally, frank uremia. The arteriosclerotic involvement of the pancreas gives all of the signs of diabetes mellitus: increased appetite, thirst, polyuria, glycosuria, and high blood sugar. The diabetic findings are more or less minimal and very amenable to treatment.

148. Prognosis.—The prognosis in arteriosclerosis depends entirely upon the functional status of the organ or organs involved. The disease is usually one of slow progress and the patients sometimes live to fairly old age. In general, however, the condition is not favorable for a long life. Death is due to the various types of end results of the diminishing arterial supply and can occur in a number of ways. Aortitis and aneurysm may cause a rupture of the aortic wall or a dissecting aneurysm. These conditions usually occur in the presence of hypertension. Coronary occlusion is usually the mode of death insofar as the heart is concerned. Cerebral hemorrhage or occlusion very frequently ends fatally, sometimes after a lingering illness. Uremia terminates the renal type. In periphal arteriosclerosis gangrene may be the mode of death.

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Embolic phenomena arising from intramural aortic thrombi frequently cause complications which result fatally.

149. Treatment.—*a.* There is no known treatment of arteriosclerosis. It is one of the accompaniments of old age and science has as yet not solved the riddle of preventing these ravages. It is generally presumed, however, that a life which has been devoid of overindulgences, excesses, and strain, is one in which arteriosclerosis is held at a minimum.

b. Such therapy as can be employed is for symptomatic relief entirely. The establishment of a cardiac regime which enables an individual to live within the limits of his cardiac and arterial reserve is the most constructive thing possible. Overexertion, overeating, and exposure to infection must be avoided. However, diet must be adequate and moderate amounts of exercise are often helpful. Drugs are of little value except when indicated for symptomatic relief. Potassium iodide has been recommended widely as an alterative, but caution is advised if this drug is employed. The treatment of the various situations arising in the local manifestations of the disease is necessarily that of the resultant condition, that is, hemiplegia, coronary occlusion, nephritis, diabetes, or occlusion of a peripheral artery. In the lodgment of an embolus in a major artery of an extremity, embolectomy is sometimes successful. Passive vascular exercises are very useful and to be highly recommended in the relief of the earlier symptoms of arterial diminution in extremities. Here the student is referred to the literature, especially that of the Pavaex therapy, for details.

150. Dissecting aneurysm.—Dissecting aneurysms are caused by a combination of weakness of the aortic wall and hypertension. This weakness is chiefly due to arteriosclerosis. The intima breaks over a point of medial pathology and the pressure of the aortic blood column begins a separation of the aortic wall in the media; such splitting or peeling of the wall involves from a small area to the entire length of the aorta. The chief symptom of this condition is pain, very excruciating and probably one of the most severe types known. There are no helpful signs. The condition is most generally missed in life. Duration of the condition is rarely more than 72 hours. Death occurs either from rupture of the aorta or from cardiovascular failure of the shock type. Very rarely, in the more minor involvements, the patient may survive. There is no treatment other than symptomatic relief, rest, and good nursing care.

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151. Recommended texts.

Diseases of the Heart (Lewis)	Macmillan.
Text Book of Medicine (Cecil)	Saunders.
Heart Disease (White)	Macmillan.

Section III

CORONARY DISEASE

	Paragraph
General	152
Coronary insufficiency and angina pectoris	153
Coronary occlusion	154
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152. General.—*a.* Disease of the coronary arteries is the principal cause of the anginal syndrome, sometimes called anginal heart failure in order to distinguish it from congestive heart failure. The chief symptom of anginal failure is pain, a severe, excruciating type of pain, of which angina pectoris is fairly typical. Coronary artery disease is of two types, coronary insufficiency, in which the blood flow through the coronaries is maintained but diminished in varying degrees, and coronary occlusion, in which the circulation through one or more of the larger coronary arteries is completely blocked. The functional result of the former is a diminished myocardial efficiency manifested by a warning angina when the limit of heart muscle activity is reached, and of the latter a myocardial infarction of varying severity with its pain, collapse (shock type of circulatory failure), and dyspnoea.

b. Disease of the coronary arteries is a part of the picture of old age and an accompaniment of senility. It is now one of the leading causes of death in the United States. In the Army in 1936 it led all other diseases (accidents excluded) as the cause of death. The condition is of especial interest to flight surgeons because it is seen more frequently in those classes subject to stress and tension, and flying personnel are notably subject to strain, both mental and physical. The cardiovascular system of flyers is subject to extra stresses imposed by the greater circulatory loads of higher altitudes, and inasmuch as persons with coronary insufficiency have a diminished cardiac efficiency, a pilot with this condition is therefore dangerous to himself and others.

153. Coronary insufficiency and angina pectoris.—Coronary insufficiency is that condition in which the blood flow through the coronary circulation is diminished to some degree by one of a number of possible causes. Angina pectoris is the most important and best known manifestation of coronary insufficiency, and will be discussed as a symptom.

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a. Etiology.—There are several causes of coronary insufficiency, the chief of which is arteriosclerosis of the coronary arteries. Other causes are incomplete blockage of the coronary ostii by either the aortic process in syphilis or by vegetations in endocarditis, spasm of the coronary arteries, or a very low diastolic blood pressure such as is seen in aortic regurgitation. Severe anemias and "shock" type of hypotension are given as adjunct causes of an insufficient blood supply to the myocardium in that in either condition insufficient oxygen reaches the heart muscle although the coronary vessels may be fairly normal. Patients suffering this condition are usually elderly, well over two-thirds of all cases occurring after the age of fifty, although the condition has been seen in young adults. Males predominate in a ratio of approximately 3 to 2. The mental workers of business and the professions seem to be most prone to the condition; laborers and outdoor workers appear to be somewhat immune. The condition is rare in the Tropics and in the colored races. Tobacco has a questionable bearing, although overindulgence is believed to precipitate attacks. As in arteriosclerosis, heredity is definitely a factor, susceptibility to the condition seeming to be inherited.

b. Pathology.—(1) The pathological conditions found in the coronary arteries vary directly with the underlying causes of the insufficiency. In atherosclerosis, the process is identical with that seen in the aorta: cholesterol degeneration, fibrosis, plaque formation, calcification. The essential difference lies in the fact that the coronary arteries are of small bore and the lumen is much more seriously encroached upon by the formation of even small sclerotic plaques. The site of the most extensive atherosclerosis is at the points of the greatest wear and tear, the anterior descending, the posterior descending, and the circumflex branches of the coronary arteries, frequency of occurrence being in that order. It is especially notable in the first portion of the anterior descending just below the ostium, and frequently that artery will be entirely free of the process except in the first 1 or 2 centimeters. In syphilis, the luetic degenerative process extends into the sinuses of Valsalva and encroaches upon the mouths of the coronaries, often diminishing the blood flow to a marked extent. Vegetations in endocarditis involve the coronary mouths in much the same manner. In these two conditions, the coronary arteries are usually free of pathology along their courses unless there is some coincident atherosclerosis.

(2) The effect of this reduced blood supply upon the heart is chiefly that of a slow degeneration. Fatty degeneration is sometimes seen. More often there are fibrotic changes, which fibrosis may be localized

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or diffuse. In the localized type, excluding infarction, there may be a small circumscribed area in which the blood supply has been diminished so severely and gradually over a long period of time (just short of occlusion) that the muscle is almost completely replaced by fibrosis. The diffuse type is more common, consisting of widely scattered areas of replacement fibrosis between muscle structures, due to a widespread but not severe restriction of the blood supply. Very often when one artery is severely involved the coronary anastomoses help make up the deficiency. In this connection, the Thebesian vessels play a prominent role and when they are especially numerous may practically take over the supply of an entire area of muscle. Recanalization of a partially occluding thrombus is a not infrequent occurrence, and this also assists in maintaining a semblance of blood supply.

(3) The result of the reduction of blood supply and the degenerative changes in the muscle is that of lowered cardiac efficiency. So long as the individual does not exceed the functional limits of his cardiac musculature and the blood supply thereto, he will have little or no trouble. However, when increased demands are made on the heart and the blood supply is insufficient to meet this increased metabolism, an ischemia develops and pain results. This pain should always be taken as a warning of myocardial difficulties, and the extra load on the heart should be immediately lessened. Another theory of causation of pain in angina pectoris is that of spasm of coronary arteries. This spasm produces a momentarily severe ischemia of the myocardium and also results in pain.

c. Symptoms.—(1) The symptoms of coronary insufficiency vary widely with the extent of the pathology and the sufficiency of the anastomoses or collateral circulation available. In the majority of cases coronary sclerosis goes unrecognized in life and at autopsy the wonder is how the individual could have lived without symptoms in the presence of so much pathology. Reports on several series of autopsies give figures ranging from 60 to 85 percent of coronary sclerosis as being unrecognized clinically.

(2) The symptoms, when found, are of two types, pain and an adjunct group. The pain is the result of the relative ischemia and is best exemplified by angina pectoris, under which heading it will be discussed. The adjunct symptoms are a miscellaneous group consisting of the manifestations of chronic nonvalvular heart disease, congestive failure in its various stages, arrhythmias, blocks, and gastrointestinal disturbances. Chronic nonvalvular heart disease and congestive failure are discussed in paragraphs 174 to 180. Arrhyth-

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mias and blocks have already been covered. The gastrointestinal manifestations are especially noteworthy in that they are at present considered to be due to reflex vagal disturbances, referred along the course of those nerves and arising in the heart. Indigestion, nausea, and vomiting, gall bladder and bowel disturbances are sometimes seen. Reference of the pain to the epigastrium, plus nausea and vomiting, often cause the condition to be confused with an acute abdomen. It should be noted in passing that the condition may be so severe as to cause sudden death; such sudden death is now considered as being due to coronary incidents other than occlusion. A partial coronary thrombosis, plus vessel spasm, is certainly possible.

d. Angina pectoris.—(1) Angina pectoris is a symptom complex manifested by substernal or precordial pain of a crushing, agonizing type. It is not a disease entity in itself. This latter view so prevalent among earlier authors has caused much confusion as to the true cause. In over 90 percent of cases diagnosed angina pectoris, some disease of the coronary arteries will be demonstrable. The remaining 10 percent must be accounted for with the theory of coronary vessel spasm. Physiologically, the pain is the result of a relative myocardial ischemia.

(2) The pain varies in intensity from mild substernal discomfort to that of greatest intensity. It is described as a choking, strangling, compressing, burning pain, rather than as cutting or knife-like. It is most commonly centered under the upper sternum. Its radiation is over a fairly typical course: precordium, left shoulder, arm, forearm, and fourth and fifth fingers. Less frequently it may radiate to the lower sternum, epigastrium, neck, face, right shoulder, arm, and back. It is so much more frequently referred to the left shoulder and arm that this distribution of the pain is sometimes considered necessary in making a diagnosis. However, if one remembers that many cases are missed because the less typical distribution is not considered, many more correct diagnoses will be made. In this connection, it should be pointed out that in the beginning the center of the pain may be at any of the points mentioned, but sooner or later it gravitates to the upper sternum or precordium. The average duration of the pain will not exceed 4 to 5 minutes. However, it may last from a few seconds to an hour. All authorities now consider that any anginal pain lasting longer than an hour is probably due to coronary occlusion. One of the best cardiologists in this country states that any pain lasting over half an hour is to be considered a probable occlusion. In frequency the attacks likewise vary widely, ranging from one nonfatal attack in a lifetime to attacks occurring at close intervals.

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(3) Attacks are induced by exertion, excitement, emotion, exposure, or even by indulgence in heavy meals. The more ease with which an attack is induced, the more severe the condition. Likewise, the more severe and frequent the attack, the more serious the outlook. Also, the more severe the attack, the wider will be the distribution of the pain. Often the pain will be intense even to the fingertips. More often the pain will be intense substernally, of lesser degree in the shoulder and arm, and be merely numbness and tingling in the forearm and fingers. Frequently, attacks are followed by short periods of parasthesias over areas in which the pain existed. During an attack rest is physiologically enforced in all but the strongest willed. It is this rest that hastens the recession of the pain, and conversely, it is the continuation of activity in the presence of the pain which frequently causes death. The fear of death during these attacks is frequently mentioned, but at present, authorities tend to discount this feature.

e. Signs.—(1) There are no signs typical of the condition. Most of those present are due to other accompanying conditions or results. The varying degrees of congestive failure present varying degrees of edema, dyspnoea, and cyanosis. Accompanying hypertension or valvular lesions will give the cardiac enlargement. Murmurs of dilatation, valve lesions, or aortitis are common. The arrhythmias, weakened heart sounds, and respiratory disturbances such as Cheyne-Stokes breathing are often seen. Gallop rhythm is an especially serious finding.

(2) There are some signs considered indicative of the condition. Sclerosis of peripheral vessels, or of the vessels of the fundus, is suggestive. However, neither of these are absolute and coronary sclerosis may exist in their absence, or may be absent in their presence. In the X-ray calcification of the coronaries may rather infrequently be demonstrated, but this very infrequency and the necessity of a specialized technique make for uncertainty. The electrocardiograph gives the nearest approach to assistance. However, here, signs are sometimes absent. The presence of blocks, either auriculoventricular or bundle branch type, as well as the arrhythmias, especially auricular flutter or fibrillation in the presence of symptoms, should create suspicion. Inversion of the T wave in leads one and two, plus changes in the QRS group, add to the evidence. However, all of these can also be attributed to other conditions, and are to be considered evidence of coronary sclerosis only in the presence of leading symptoms.

f. Prognosis.—(1) The prognosis is at best unfavorable. The condition is usually progressive, leading to either disability or death.

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The postponement of disability or death depends in a large measure upon the patient, whether or not he is willing to live within the limits of his cardiac capacity. In many cases, a reasonably comfortable and prolonged existence can be had by the observance of a properly laid out regime.

(2) In spite of the best medical advice, however, most patients suffering appreciable degrees of coronary insufficiency will be cardiac cripples. Fear is the greatest factor here, with actual limitation of activity caused by pain a very close second. The insufficiency increases usually very slowly to reach one of several possibilities, coronary occlusion, congestive failure, or death. Death during an attack of pain is due to a reflex cardiac standstill, and is usually very sudden and dramatic. Death other than in an attack may occur from coronary occlusion, congestive failure, cerebral accidents, renal complications, or intercurrent infections. In angina pectoris, the average outlook for duration of life after onset is 5 years. This may vary, however, from a few days to as many as 20 years.

(3) There is no way to predict duration of life. The prognosis in general varies with the degree, duration, and transmission of pain. The more severe these factors are, the more serious the outlook. Ease of induction of attacks and frequency of occurrence also have a bearing. The easier attacks are brought on and the more frequently they occur, the more severe the disease and the shorter the prospect for duration of life. The presence of other cardiac conditions or systemic diseases likewise adversely influences prognosis. The more nervous and excitable the patient the shorter his life is apt to be. It should be noted in passing that the onset of congestive failure, auricular fibrillation, or a healed coronary occlusion more frequently than not abolishes all anginal pain.

g. Treatment.—(1) The treatment of coronary insufficiency resolves itself into the treatment of pain and the prevention, insofar as possible, of recurrence of attacks. The treatment of an attack further resolves itself into two factors, rest and medication. Rest in most cases is involuntary and life-saving. The quick acting nitrites are specific for the relief of true anginal pain. Amyl nitrite in 2 or 3 minim pearls for inhalation, or nitroglycerin in 1/100-grain tablets for sublingual use, are the drugs of choice. Both act very rapidly, in a matter of seconds, and are truly remarkable for their efficacy in the relief of an acute attack. In the prevention of attacks, several factors arise. These are rest, diet, limitation of activity, medication, and surgical procedures. Rest, diet, and limitation of activity are the most useful and the most important. It is essential that every patient

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be placed on a regime fitted to his cardiac capacity. The amount of rest required depends on the severity of attacks and ease of induction, and must be determined individually in each case. Diet should be adequate, but easy enough to digest so that attacks will not be induced from the gastrointestinal tract. In this regard, it is often very helpful to use six or more small meals per day, rather than three larger ones. Activity should be limited and slowed to the point wherein attacks will not be precipitated by exertion. Here again each individual case requires study in determining this point. Drugs should be used when these methods are not entirely effective. Coronary dilators are given over long periods of time in an attempt to maintain an increased coronary flow. Theophylline ethyline diamine and the theobromine salts are the coronary dilators of choice. Theophylline ethyline diamine is sold under a variety of trade names, the best known of which is aminophyllin, and should be used in 1½-grain doses four times daily over long periods of time. It is often well to alternate this drug in 20- or 30-day periods with the theobromine salts, theocalcin, or diuretin, the dosage of which is 15 grains t. i. d. A combination of plain theobromine in 5- to 10-grain doses with luminal has proved very satisfactory as an alternative to aminophyllin, in that the luminal both depresses sensitivity and the unpleasant side reactions of theobromine. The nitrites should be used prophylactically in such instances where necessary, but attack provoking activity must be indulged in. Here nitroglycerin in 1/100-grain doses or sodium nitrite in 1-grain doses, taken 3 or 4 minutes before the effort, will often avert the attack. Erythrol tetranitrite is not recommended because of the cost of the drug and the fact that it has few, if any, advantages over the two mentioned. Sedatives are useful in nervous individuals and serve to lessen apprehension somewhat. Phenobarbital or bromides best serve the purpose here, in that they can be given over prolonged periods.

(2) Surgical procedures in general are unsatisfactory. Nerve injection and nerve resections have been tried but are not universally successful, and have the additional disadvantage of removing the patient's warning signal (angina) of impending trouble. Subtotal thyroidectomy is successful only in the presence of hyperthyroidism and is not to be recommended in routine cases. The best surgical procedure yet devised for the relief of pain is transplantation of a new blood supply to the heart muscle. This is accomplished by grafting subpectoral muscle pedicles, or omentum pedicles, through the diaphragm to the heart muscle itself, subpericardially. Other experimenters have used diaphragmatic pedicles. The operation, however, is yet too new to evaluate its efficacy, but seems to hold out the greatest

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promise of constructive treatment yet devised. It should be noted that many other procedures have been tried, but none are of any proved value.

154. Coronary occlusion.—Coronary occlusion is that condition in which the flow of blood through one of the coronary arteries or branches is completely blocked off. The end result is death of the cardiac area (distal to the blockage) which is ordinarily supplied by that vessel.

a. Etiology.—The etiological factors to be considered in coronary occlusion are essentially those noted in coronary insufficiency. Occlusion in most instances is merely insufficiency climaxed by complete shutting off of the blood flow. The greatest cause of occlusion is a thrombosis arising on an arterio-sclerotic process in one of the arteries. Complete blockage of the ostium of either artery by syphilis or an endocarditic process is another cause. Embolism is a third cause. The latter two are relatively rare, thrombosis being so predominant that coronary occlusion is very often referred to as coronary thrombosis. The condition is very common and is now the leading cause of cardiac death. The age group involved is somewhat older than that seen in coronary insufficiency, the condition being rather rare before 40 and relatively common after the age of 50. Occasion occurs more frequently in the presence of hypertension or diabetes than in their absence. There is no precipitating cause known, although it was formerly thought that excitement, emotion, and exertion brought on the attacks. However, recent statistical reports have shown that the attacks occur just as commonly at rest as during activity, in fact, occlusion occurring during sleep is rather classical. This concept that patient activity just prior to the pain of occlusion is coincidental rather than causative is based on the concept recently advanced that the formation of coronary thrombi is gradual, often requiring several days before resulting precordial pain is produced, the symptoms and signs produced being merely the final phase of a process initiated some time previously. It is to be emphasized that occlusion is very commonly preceded by attacks of angina pectoris and that except in the case of embolism coronary insufficiency is the predecessor to all occlusion.

b. Pathology.—(1) The pathology of coronary occlusion is essentially that of infarction of the area normally supplied by the vessel occluded. The process is one of necrosis of myocardium, endocardium, and pericardium. Very small areas of infarction will involve the myocardium alone, larger areas will include the endocardium, and still larger areas will also include the pericardium. Sterile

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thrombi form over the areas of endocardial and pericardial necrosis, and it is from these endocardial thrombi that the peripheral emboli often seen in this condition arise. The necrosis proceeds to one of two terminations, healing or rupture. Healing is by fibrosis and the end result in an area of dense fibrotic tissue entirely replacing the destroyed cardiac structures. Frequently, calcium is deposited in this fibrous area. Rupture, which occurs most frequently during the second week, results in massive hemorrhage and sudden death. It is interesting to note that the strong fibrous parietal pericardium is not involved in the infarction and often prevents rupture by being plastered to the heart wall by the sterile visceral pericardial thrombus, thus lending its support to the weakened wall. The entire infarctive process may be cut short at any stage by death either from cardiac failure or from shock. The size of the infarct (usually 3 to 6 centimeters in diameter) and the extensiveness of the destruction depend upon the size of the artery at the point of occlusion and the amount of collateral circulation available. The location of the infarct depends entirely upon the point of occlusion. Two locations are most common, anterior apical and posterior basal. The anterior apical type is the result of occlusion of the anterior descending branch of the left coronary, sometimes called the artery of sudden death. Involvement is usually within the first 2 centimeters of that branch and causes infarction of the left ventricular wall near the apex. The posterior basal type is the result of occlusion of either the right coronary artery or the circumflex branch of the left. Infarction here is usually seen in the right ventricular wall near the base. Extensive infarctions of either type will involve the interventricular septum and possibly small areas of the other ventricle. The anterior apical type is slightly more common than the posterior basal although it was previously thought that the former was markedly predominant. When smaller branches or arterioles are involved, the infarction is correspondingly small, atypical, and frequently silent.

(2) It should be remembered that when sudden, immediate death is the result of coronary occlusion, no infarctive process will be seen. The suddenness of death does not allow the necessary elapsed time to produce necrosis, and at autopsy a normal looking myocardium will be seen, often necessitating a close search in all coronary branches for the occlusion.

c. Symptoms.—The three main symptoms of coronary occlusion are pain, collapse, and dyspnoea. Any or all may be absent or they may vary in degree from very slight manifestations to those of extreme severity. The typical pain of an acute, severe occlusion is substernal in

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location and radiates to the neck, head, left shoulder and arm, much as does the pain of angina pectoris. It also has the atypical distribution noted with angina pectoris, and it is not uncommon to have the pain centered in the epigastrium. The pain is usually severe, agonizing, boring, crushing, pressing, or burning in type. It is persistent, continuous, unfluctuating, and lasts intensely for several hours, gradually receding over a period of several days. Failure of nitrites to relieve the pain is very characteristic of occlusion. The collapse is of the shock type, often being severe enough to cause early death. Sweating, restlessness, fear, weakness, or even prolonged coma are commonly seen. The dyspnoea is of all degrees, to the severest gasping. Cheyne-Stokes type of respiration is common. Other lesser symptoms are also to be noted. Nausea, vomiting, diarrhea are to be attributed to reflex activity of the gastrointestinal tract. Dizziness, fainting, syncope, convulsions are due to reflex activity of the nervous system. Nervousness, agitation, restlessness are also seen. Embolic phenomena may become manifest during the first 10 days in any of the systems or extremities. Emboli to the lungs are common when the right ventricle is involved. Emboli from the left ventricle most commonly lodge in the extremities, kidneys, spleen, mesenteric arteries, or brain. Congestive failure with its dyspnoea, cough, edema, and ascites are frequently in evidence. Onset is sudden, often dramatic. Sometimes there are premonitory pains of varying severity, or the patient reports that he has not felt entirely well during the preceding 12 or 24 hours. It is to be emphasized that sometimes occlusion does occur which is symptomless, or so atypical that it will be missed entirely and found only at autopsy.

d. Signs.—(1) The signs to be noted in occlusion are numerous and vary widely in the number and severity of their occurrence. Often many are absent or very slight and one or two predominate the picture. Most of the signs as noted below can be attributed to the accompanying shock, cardiac dilatation, or to the formation of the infarct. The signs to be noted are shock, tachycardia, low blood pressure, weakened cardiac sounds, dilatation murmurs, friction rub, breathlessness, fever, leucocytosis, increased sedimentation rate, arrhythmias, blocks, electrocardiographic changes, embolic manifestations, and later congestive failure. The shock and collapse are typically noted in the pale, haggard, anxious, agonized expression and in the profuse sweating. The ashiness is very typical and is sometimes complicated by cyanosis. The pulse is rapid, weak, small, often impalpable. The tachycardia varies from 100 to 150, rarely above that in the absence of arrhythmias. The blood pressure drops markedly in practically every case, and systolic

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readings below 80 are not uncommon during the first few days. (It is to be remembered that during angina pectoris attacks, the blood pressure rises.) Cardiac sounds are weak, distant, of very poor quality, and often tick-tack in type. Dilatation is very frequently present with its apical and basal murmurs. When the interpulmonic pressure rises, murmurs appear at the pulmonic areas. When the pericardium is involved, a transient friction rub may be heard; but its absence is not to be taken as indicating the absence of pericardial thrombus formation. Breathlessness is very common and, as noted under symptoms, the dyspnoea varies from the slightest to Cheyne-Stokes breathing. Fever appears 12 hours or more after the occlusion and rises to between 101° and 102°. This gradually recedes and is usually gone within a week. A leucocytosis of 12,000 to 20,000 appears shortly before the fever, but in general follows its course. Both are due to the inflammation accompanying the myocardial necrosis, and subside with the healing process. The sedimentation rate is increased, corresponds roughly in degree to the leucocytosis, but lasts longer. It is worth noting that a sedimentation rate which is still normal up to 10 days after substernal pain is almost in itself justification to rule out infarction. Arrhythmias appear, varying from occasional extrasystoles to paroxysmal tachycardia and auricular fibrillation. Heart blocks of varying degrees appear when the infarction is extensive enough to involve any portion of the conduction system. Gallop rhythm or a slow rate is the usual manifestation of heart block.

(2) Probably the most significant changes occur in the electrocardiogram. Even here, however, little or no change may be manifest. In the classical leads an anterior apical infarction is indicated by a Q1–T1 pattern and a posterior basal infarction is indicated by a Q3–T3 pattern. The chest leads give their greatest amount of information in this condition. Blocks or arrhythmias will distort the patterns. Electrocardiograms should be taken serially at 24 to 48 hour intervals to render the greatest amount of information, because the progressive changes in necrosis and healing are best followed through this instrument. For more detailed information concerning electrocardiograph patterns in this condition, the student is referred to the standard texts. Embolic manifestations occur in the first few days of the disease and give signs typical of the location of the newly occluded vessel. Congestive failure with its multiplicity of signs may be either an early or a late manifestation. All fourteen of the signs listed are of importance, and although several may be absent in any given case, all should be searched for and considered before arriving at any definite conclusion.

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e. Prognosis.—(1) The prognosis at best is poor. It is very difficult to even guess the outcome in most cases. Those appearing most hopeless often recover remarkably, and those appearing mild often die suddenly or are left with considerable disability. The outlook after occlusion is either early death or a life of cardiac invalidism lasting a few years. Death may occur suddenly in the first few days from shock, ventricular fibrillation, or complete heart block. If the initial shock is survived, death can then occur from intercurrent infection, congestive failure, or renal insufficiency. If recovery from the acute condition occurs, the average length of life is 2 to 3 years, although with good luck and a proper cardiac regime it may be extended much beyond that. Duration of life from 15 to 20 years has been reported at intervals in the literature. At best, if the acute attack is recovered from, the patient will be a cardiac cripple and forced to markedly restrict activity, unless he is one of those lucky individuals who seem to recover remarkably well and are able to resume practically full pre-attack activity.

(2) There are several signs which in general indicate a poor prognosis. Gallop rhythm, pulsus alternans, ventricular tachycardia, heart block, embolic phenomena, congestive failure (especially edema of the lungs) are those of the most importance. Any evidence which points to large areas of infarction also emphasizes the seriousness. Severe grades of shock are bad prognostic signs. Any coexisting cardiac disease, especially hypertension, makes for a more serious outlook. In general, the younger the individual suffering the attack, the more hopeful the outlook for immediate recovery and for length of life following recovery.

f. Treatment.—(1) Treatment is to be directed at the attainment of rest, absolute and complete, physical, mental, and emotional. To attain this the patient should be kept in bed and not moved for the first few days. Good nursing care is essential in attaining this objective. Nothing should be given by mouth for the first 72 hours. Morphine in quantities sufficient to overcome pain and alleviate shock and restlessness should be given. Doses of ¼-grain every half hour until 1 grain has been given are not excessive; this to be followed with enough more of the drug at appropriate intervals to maintain the effect. Nitrites are useless in the relief of pain. In cases of severe shock, cyanosis or dyspnoea oxygen administration should be carried out, preferably by means of a tent. Oxygen administration relieves considerable of the burden placed on the heart under the above conditions. Glucose intravenously is helpful in maintaining nourishment and combating shock. Fifty cubic centimeters of glucose can be given

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per day in solutions varying from 10 to 50 percent, depending entirely upon whether a secondary objective of fluid administration or of diuresis is desired. Caffiene sodium benzoate in 7½-grain doses intramuscularly will aid in supporting respiration. A pharmaceutical house product, coramine, used for respiratory support, has been used successfully in some cases. In the earlier phases of pulselessness, adrenalin is sometimes given, but there is some disagreement at present as to its advisability. In the presence of Stokes-Adams syndrome adrenalin is sometimes life-saving. In ventricular tachycardia quinidine sulphate in 5-grain doses is indicated. If the patient is dehydrated, fluids should be given by rectum, preferably, but very slow, intravenous administration is sometimes recommended. The measures as outlined above for an acute attack should be carried out, at least for 72 hours, possibly longer, with slight alterations if necessary.

(2) Following survival of the critical period the treatment should be modified considerably. Bed rest should be maintained, for from 4 to 8 weeks, or longer if necessary. Then 2 weeks should be spent in getting the patient gradually out of bed, increasing the degree of activity daily until the patient is able to be up and about for a few minutes at a time. Morphine should be stopped and a milder sedative substituted. Bromides, chloral, luminal, and sodium amytal are useful in this respect. Aminophyllin should be begun in 1½-grain doses four times per day and continued indefinitely. Diet should be at first liquid, gradually increasing the caloric and solid content until the patient is receiving adequate nourishment in a normally appetizing form. Some authors have recommended low caloric diet over long periods with the object in mind of reducing bodily metabolism. This regime has its points and should be considered. Good nursing care is essential throughout, in order to relieve the patient of all unnecessary exertion and to maintain hygiene.

(3) After the patient is up and about, the regime established should be one suited entirely to the limits of his cardiac capacity. From 3 to 6 months should be consumed in resuming the fullest activity possible under the new cardiac status. It is to be emphasized that the patient's cardiac reserve has been reduced by the infarction, and that even though there is apparent full recovery caution should be observed in the matters of exercise, exertion, diet, and intercurrent illnesses. The more the patient will cooperate in the new regime, the greater are his chances of extending his life over the 2-year average outlook. In many cases, congestive failure will intervene or the patient will live on the verge of failure. Here

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digitalis is indicated, and the patient should be treated as any other congestive failure. It will be noted that nothing has been said about the use of digitalis in the acute phase. Digitalis is definitely contraindicated unless there is evidence of congestive failure. In an average coronary occlusion congestive failure will not become manifest until after the first few weeks.

155. Recommended texts.

Disease of the Coronary Arteries and Cardiac Pain (Levy)	Macmillan.
Heart disease (White)	Macmillan.
Clinical Heart Disease (Levine)	Saunders.
Diseases of the Heart (Lewis)	Macmillan.
Failure of the Circulation (Harrison)	Williams and Wilkins.

SECTION IV

HYPERTENSION AND HYPERTENSIVE HEART DISEASE

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156. General.—*a.* Hypertension is that condition in which the arterial blood pressure rises above readings ordinarily accepted as normal. Early in the course of the disease the elevations of pressure are transient and occur at wide intervals, whereas later the blood pressure tends to become permanently high. The condition is of great importance in that it is one of the four leading causes of heart disease. The Army attaches much significance to a finding of even mild hypertension in cadet applicants, because it is considered an indication of future trouble. This assumption is based on the fact that future hypertensives frequently have slight transient elevations of blood pressure when placed in situations of emotional pressure, such as cadet examinations. Such transient rises in blood pressure are considered by some authorities as fairly reliable indications of future hypertension.

b. Hypertension and hypertensive heart disease cannot be separated in a discussion of this type. The former is necessarily the

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cause of the latter. Usually there is an interval of approximately 10 years between cause and effect. The close association between hypertension and arteriosclerosis should also be mentioned. The two conditions coexist in a large percentage of advanced cases, and although hypertension is considered a cause of arteriosclerosis, arteriosclerosis is not necessarily a cause of hypertension. Hypertension of any degree will not exist for long without producing thickening of the arteries, but arteriosclerosis may exist for years without a rise in blood pressure. However, arteriosclerosis does increase hypertension somewhat by increasing peripheral resistance to blood flow.

157. Etiology.—*a.* There is no single cause of hypertension known. Many theories exist, too numerous to mention in detail. A rough idea can be gained from the following general list: mechanical factors of peripheral resistance, toxins, blood chemistry alterations, dietary errors, obesity, allergy, endocrine disturbances, emotional upsets, nervous and physical strain, foci of infection. All may have a bearing but no one theory adequately explains the situation. The most acceptable theory is that for some unexplained reason the arterioles become irritable, vasospastic constriction sets in and peripheral resistance is increased. To overcome this increase in peripheral resistance, the heart must increase its hydrostatic force, which is the blood-pressure level.

b. The most acceptable division of hypertension into types is primary (essential) and secondary (organic). Eighty-five percent of hypertension is of the primary type, of which no cause is known. The remaining 15 percent is of the secondary type, for which seven possible causes are listed: coarctation of the aorta, glomerular nephritis, tumors of the suprarenal gland, hyperthyroidism, arteriosclerosis, traumatic arteriovenous fistula, and aortic heart disease. It is not universally agreed that arteriosclerosis is a cause of hypertension.

c. As noted above, hypertension is one of the leading causes of cardiac disability. It leads most of the lists published by various authors which give the frequency of occurrence of causes of heart disease. All are agreed that it causes at least 25 percent of heart troubles, and figures as high as 40 percent have been given. It is a disease of persons past middle age, occurring chiefly in those of fifty and over. Less than one-fourth of the cases of permanent hypertension occur in individuals under fifty. The distribution between the sexes is just about equal, although one authority states that females predominate in a ratio of 3 to 2. Climate seems to have some effect in that the condition is seen much less frequently in the

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Tropics. Likewise, it is seen less frequently in some races, namely, the Chinese and the African Negroes. White points out that, although this is true in the African Negro, the incidence of hypertension in the American Negro race very closely approximates that of the white race.

d. Heredity and emotional make-up seem to be the two etiological factors of the most importance. Here, as in arteriosclerosis, there is a definite familiar tendency toward the condition. The suceptibility to hypertension can be traced back through generations. In almost every case of hypertensive heart disease, careful questioning will elicit a family history of similar troubles. An emotional make-up of the easily disturbed type, the other predisposing factor of importance, is also an inherited family trait. As noted above, those people who, when put under emotional stress such as meeting a new situation show transient rises in blood pressure, are those who later become the permanent hypertensives. This transient rise in blood pressure is very frequently seen during original cadet examinations. So much importance is attached to this factor that the Army has recently added a finding of "unstable blood pressure" as a disqualification for appointment as flying cadet.

e. The cause of hypertensive heart disease, is, of course, hypertension. The etiological factors are the same as for hypertension, except that the cardiac changes become manifest only after the hypertension has existed for a period of years. Approximately 90 percent of hypertensive heart disease is caused by the essential type of hypertension.

158. Pathology.—*a.* There are no pathological changes entirely characteristic of the disease. All of the findings to be noted are the result of long continued high pressure levels which produce their changes chiefly in the arteries, the aorta, and the heart. The arteries and arterioles lose their elasticity, become thickened, sclerosed, and suffer diminution in the size of the lumen. The result is a lessening of blood supply to the periphery of the body, such lessening being especially prominent in kidneys, pancreas, and liver. The aorta undergoes a dynamic dilatation, seen during life by fluoroscopy and X-ray. However, at autopsy this dilatation is very difficult to detect, inasmuch as it is a dynamic phenomenon rather than an organic thinning of the aortic wall. The heart becomes hypertrophied as a result of the increased burden placed upon it. This hypertrophy is predominantly of the left ventricular type. The heart very frequently weighs between 500 and 800 grams. Simple hypertrophy does not exist for long, however, because as the strain

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upon the left ventricle continues, congestive failure sets in and that ventricle dilates. As the degree of failure increases, the left auricle also dilates, the pulmonary pressure then rises and the right side of heart ultimately becomes involved. Death by congestive failure is one of the principal modes of termination of this disease.

b. The combintion of very high pressure levels and severe arteriosclerosis has a very definite set of possible end results. In the arteries of the brain, cerebral hemorrhage predominates, although a group of manifestations aptly called hypertensive encephalopathy frequently coexists or precedes the hemorrhage. In the aorta, dissecting aneurysm is made possible. In the heart, the degenerative valvular changes, especially sclerosis of the aortic valve, complicate the picture and add to the burden of the heart. The occurrence of coronary sclerosis is seen in a large number of cases and the diminution of the blood supply to the hypertrophied heart wall hastens the occurrence of congestive failure. All of the other systems of the body likewise must be affected, but none present characteristic groups of findings such as those listed.

159. Symptoms.—*a.* There are no symptoms which can be definitely ascribed to the elevation of blood pressure in itself. Such symptoms as are present are usually due to the onset of complications, and it is almost an axiom that most hypertensives will be symptomless so long as the cardiac reserve is able to carry the added burden, or until arterial complications occur. Most individuals with hypertension feel well, lead active lives, and are apparently in good health. The high blood pressure is usually discovered at some routine examination such as insurance, annual physicals, or during coincident examination at the time of other illness. In many cases, once the patient has become aware that high blood pressure levels exist, a neurosis sets in, due to the fear of the condition, which has been engendered by widespread knowledge of the disease and its implications. Very frequently this neurosis is much more difficult to deal with than the actual symptoms of the organic changes.

b. When symptoms do occur, the onset will be very gradual and it will be almost impossible to determine their actual beginning. Fatigue, restlessness, dizziness, loss of energy, headache, and nosebleed are often seen in the earliest phases. The headache is fairly typical and varies from a dull, occipital or frontal ache to severe throbbing paroxysms. The headaches usually occur after exercise, exertion, or when the patient remains long in the reclining position. Nosebleeds are fairly common, spontaneous in type, usually small in amounts. When the heart begins to fail, early breathlessness, slight

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evening swelling of the ankles, and nocturnal dyspnoea are manifest. Palpitation, precordial aches, and often severe angina become annoying on exertion. Later in the progress of the disease, all of the signs and symptoms of advanced congestive failure occur if the case is to reach its termination in that manner. If the termination is to be reached by the cerebral or renal routes, the manifestations of those complications will be seen.

160. Signs.—*a.* The only definite sign of hypertension is a blood pressure reading above the figures accepted as the maximum of normal. Normal is usually accepted as from 110 to 130 millimeter mercury systolic, and 70 to 85 millimeter mercury diastolic. The borderline readings usually considered as abnormal are 160 systolic and 100 diastolic. Hypertensive blood pressure readings will vary anywhere between these borderline figures and 300 systolic to 175 diastolic. Even higher findings have been reported. The Army accepts 150 millimeter mercury as the maximum normal systolic reading, and 100 millimeter mercury as the maximum normal diastolic reading. For persons under 25 years of age, 140 millimeter mercury is the maximum systolic reading acceptable. For flying cadet applicants a systolic pressure of 135 or more or a diastolic pressure of 90 or more is disqualifying.

b. Single blood pressure readings are of no value unless well within normal limits. Blood pressure is an extremely variable phenomenon. It is markedly influenced by excitement, emotions, exertion, rest, and fatigue. Even under equivalent conditions, it varies from time to time in the same individual. Identical readings between the two arms are practically impossible to secure, although the variation here will not be great. Blood pressure varies with the size of the vessel compressed, as illustrated in the difference between arm and leg readings. Further, some individuals have blood pressure "storms" or crises in which the blood pressure for some unexplained reason will become high and remain high for variable periods of time, eventually returning to normal. Therefore, a series of blood pressure readings should be taken before arriving at any conclusions as to the status of hypertension in any given case. The Army requires that "when the blood pressure estimation at the first examination is regarded as abnormal, or in case of doubt, the procedure will be repeated twice daily (in the morning and in the afternoon) for a sufficient number of days to enable the examiner to arrive at a definite conclusion."

c. Basal blood pressure is that pressure maintained in the body when the influences and control of emotions or the conscious mind are removed. This is determined during sleep, under anesthesia, or

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under marked sedation. This level may be determined by giving the patient three doses of sodium amytal, gr. III, at hour intervals, following a preparation of 24 hours' rest. Following the effect of the drug, an accurate basal reading can be secured. Often basal readings will be normal, whereas in the waking state blood pressure will be abnormally high. Cases in which the basal pressure returns to normal or near normal carry a good prognosis, whereas those cases maintaining high levels under anesthesia or sedation have much poorer outlooks. These should be accepted as having permanent changes or a fixed hypertension.

d. The signs of hypertension seen in the heart prior to onset of congestive failure are two in number, left ventricular enlargement and an accentuated aortic second sound. The enlargement of the left ventricle is the most constant and most important, being present in practically every case of hypertension of any degree which is of long enough duration to produce cardiac changes. Very early in the onset of failure, dilatation of the left ventricle and aorta occurs, giving rise to systolic murmurs in the basal and apical areas. As the failure progresses, dyspnoea, cyanosis, edema, and anasarca are added to the picture. Later, gallop rhythm, pulsus alternans, arrhythmias, and blocks will be seen. As congestive failure runs its cycle, the blood pressure levels off and the systolic pressure often reaches normal or near-normal levels. However, the diastolic pressure does not follow the systolic down and remains at higher levels such as 110 to 120 millimeters mercury. This fact is of considerable importance in determining etiology in those cases of congestive failure not seen prior to the actual failure.

e. The retina furnishes a very important group of findings in hypertension. Early in the condition there are often arteriolar spasms of various degrees. Later, retinitis with hemorrhages, exudates, and edema of the disk will be seen. This finding is of such constancy that the Mayo Clinic group classify all essential hypertension cases into four grades on the basis of retinal findings. These groups in order of severity are—

(1) *Group 1.*—Slight to moderate increase in blood pressure which ordinarily becomes normal as a result of rest. Mild sclerosis of retinal arteries.

(2) *Group 2.*—Moderate to severe hypertension. Moderate sclerosis of retinal arteries. Occasionally venous thrombosis and arteriosclerotic retinitis.

(3) *Group 3.*—Moderate to severe hypertension. Angiospastic retinitis.

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(4) *Group 4.*—Severe hypertension. Angiospastic retinitis, edema of optic disks.

This grouping has been found extremely useful in the clinical classification of hypertensives and in the evaluation of prognosis and is offered on that basis only.

f. X-ray and electrocardiographic findings in hypertension are not at all typical, and both are due to complications rather than to the disease itself. In the X-ray, left ventricular hypertrophy of the concentric type is seen at first, followed later by general enlargement and dilatation. Dynamic dilatation of the aorta is a fairly frequent finding and determined best by fluoroscopy. In the electrocardiograph is found left ventricular preponderance, ventricular complex alterations due to myocardial changes, the various degrees of bundle branch block, and arrhythmias. Marked cases of long standing hypertension sometimes show a definite pattern consisting of left axis deviation, inversion of T_1 and T_2, depression of RT_1, compensatory elevation of RT_3 with diminution of Q_4 and T_4. The finding of this pattern in its various degrees denotes cardiac changes of a serious nature.

g. Extracardiac complications of hypertension are chiefly cerebral and renal. Cerebral complications will be manifested by the various neurological changes seen in paralyses, parathesias, reflex abnormalities, and sensory disturbances. Early renal changes are manifested by increase in night volume of urine to the point where day and night volumes are practically equal. This produces nocturia, which is very disturbing and leads to considerable loss of sleep in patients who otherwise ordinarily sleep poorly. As the condition progresses, the specific gravity differential between day and night urine levels out and tends to become lower generally. Following these evidences of renal impairment, the early changes of renal insufficiency, nitrogen retention, and ultimately clinical uremia are seen.

161. Prognosis.—*a.* Hypertension is a progressive condition, beginning insidiously in middle life and advancing slowly to death in from 10 to 20 years later. Manifest heart disease is seen on an average of 10 years after the onset of hypertension. Death occurs most commonly from one of three conditions, congestive failure, cerebral hemorrhage, or uremia. This triumvirate of death is sometimes superseded by two other possibilities, intercurrent infection, which accounts for a large number of deaths, and anginal failure, which accounts for a few. Anginal failure is seen only in the presence of the arteriosclerosis which complicates a large proportion of cases occurring in older individuals. Coronary sclerosis, especially, adds to the burden

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of the heart, and if it is not the cause of anginal failure, hastens the occurrence of congestive failure.

b. The individual prognosis in hypertension is for a 10- to 20-year duration of life, the last few years of which will be more or less as an invalid. Certain signs render the prognosis much more grave. The more youthful the individual at onset, the more serious the disease is apt to be. The presence of angina or of congestive failure is ominous. Congestive failure in hypertension is a terminal condition and usually progresses with few or no periods of improvement to death within a year. The presence of cerebral or advanced renal signs carries its own serious outlook. Very high pressure readings are serious. Sustained high systolic pressures of 220 and above, or sustained diastolic pressures of 125 and above, have a most serious prognostic significance. Marked retinal changes of the Mayo group 4 type means an outlook of perhaps not more than 6 months. Pulsus alternans, gallop rhythm, and paroxysmal dyspnoea usually indicate death within 2 years. A very marked enlargement likewise carries a serious outlook. On the other hand, the less serious signs, not mentioned in this paragraph, should give a more or less hopeful forecast, in that the individual who is carrying on his full duties without discomfort or signs of cardiac involvement is apt to give many more years of service before disability. The question of flying with a hypertension should be settled by permitting flying duty in those cases of milder hypertension who show no cardiac changes; but once cardiac changes or any of the serious signs noted above appear, flying duty should be terminated.

c. The term "malignant hypertension" is often seen in the literature. This term is used to designate those cases of hypertension in which the course is rapidly fatal and death often a matter of only a few months from the time of onset. Sometimes a slowly progressive case will suddenly become very rapidly worse and proceed to an early death. These and other serious cases are termed malignant. The term is merely a name used to denote seriousness and is not one which denotes a definite clinical entity such as essential hypertension. Very few cases of hypertension will fall into this classification.

d. It will be noted that up to the present time no attempt has been made to separate organic from essential hypertension in the discussion. Except for one or two features to be mentioned, they are practically the same insofar as signs and symptoms are concerned and vary only slightly in prognosis. However, each of the seven organic causes has its own characteristics and prognosis, and thus the prognosis of the resultant hypertension is somewhat af-

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fected by the characteristics of the causative disease. It should be noted that all except arteriosclerosis are conditions of younger individuals and that, further, arteriosclerosis is not universally recognized as a cause of hypertension. Also, subtotal thyroidectomy very frequently relieves the hypertension and the individual proceeds from that time on in a fairly normal cardiovascular condition. Glomerular nephritis is the most common organic cause of hypertension and is rather rare after the age of forty, whereas essential hypertension is somewhat rare before that age. Likewise, in glomerular nephritis the blood pressure rises gradually, assumes a lower level, and maintains that level much more steadily than does the essential type. The urinary findings of albumin and red cells help make the differentiation. Most of the renal nephritics will have died a uremic death before the average essential hypertension is much beyond the early fluctuating stage. Although essential hypertension cases do die uremic deaths, they usually do so at a much older age than the organic type. For more complete discussions of the possibilities of each of these conditions, the student is referred to the standard texts.

162. Treatment.—*a.* The treatment of hypertension is divided roughly into two phases, the care before and the prevention of cardiac failure, and the treatment of congestive failure and other complications when they do occur. The care before failure is directed at relieving the load on the heart, in other words, living within the limits of the cardiac capacity. This is accomplished by one or both of two methods, medical and surgical. The medical regime consists of rest, diet, and medication. Rest is all important, for with this procedure alone life can be extended for years. Hypertensive individuals should have from 8 to 10 hours' sleep daily and a nap each day if possible. In most cases it is not necessary that they give up work, but that their work be continued at a more moderate pace commensurate with the amount of sleep indicated. Persons subject to strain in their work should take regular and frequent vacations, week ends, and days off, as needed. The value of sojourns at spas cannot be overestimated. While rest should be directed at relieving the load on the heart, exercise should not be neglected. Appropriate amounts of nonstrenuous exercises are to be advised in those cases in which the cardiac reserve will tolerate it. Diet should be limited to gradual reduction of overweight or to the building up of run-down constitutions. Reduction of weight alone in the overweight will often reduce blood pressure readings appreciably and serve to prolong life indefinitely. Formerly it was

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thought that protein and salt should be restricted. More recent opinion holds that protein and salt are not harmful if indulged in in normal amounts, and that except for weight reduction or for weight gain, diet should be entirely normal. Excesses in alcohol, tobacco, tea, and coffee are to be discouraged. Medication holds little hope except in the relief of symptoms. Practically every type of drug and physiotherapeutic procedure has been tried at one time or another as a cure, and all have met with universal failure. Sedatives, especially in the form of luminal in small doses, seem helpful. Analgesics should be used for pain, especially the headaches. Aminophyllin is sometimes helpful in the anginal cases. Thyroid extract in doses of 1 to 3 grains per day may be tried. Potassium iodide used as an alterative in from 3- to 10-grain doses t.i.d. sometimes seems helpful. Where symptoms are particularly bothersome and the pressure is high, as in the hypertensive "storms" mentioned above, measures may be taken to lower the pressure 10 to 20 points. Potassium sulphocyanate in 1½- to 3-grain doses t.i.d. is often effective here. Larger doses are not recommended, nor is prolonged usage, because of the toxic effects of the drug. Intravenous magnesium sulphate, ¼-grain per pound body weight in a 2½-percent solution of triple distilled water will likewise relieve symptoms. The old-fashioned method of bleeding still has its uses in this type of case and especially in those cases where pressures become malignantly high and cerebral accidents seem imminent. From 200 to 300 cubic centimeters of blood removed from one of the veins will usually tide the individual over the danger period.

b. The treatment of congestive failure when it occurs in the presence of hypertension is in no way different from that indicated in congestive failure from other causes. Likewise, the treatment of the complications other than congestive failure varies little from that ordinarily indicated, except that frequently there is considerable advantage to be gained by lowering the blood pressure. In congestive failure the weakening myocardium itself takes care of this factor.

c. Surgery is useful in two conditions, the removal of foci of infection and in nerve resections. The removal of foci of infection may cause more or less permanent recession of high pressure levels in many cases. Removal of foci should be a routine procedure in all cases whose condition will enable them to withstand successfully the type of surgery indicated. Nerve resections are now being done in some of the larger clinics on selected cases, those in which the

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permanent systemic changes which result from hypertension have not yet occurred. Specifically, one clinic requires that cases for subdiaphragmatic sympthectomy must meet three conditions: first that the hypertension be progressive; second that heart and kidney function be good; third that blood pressure returns to normal or near normal under sodium amytal or sleep. Although the procedure is yet somewhat radical, results have been good and the operative risk not above normal for major surgery. The result is a permanent vasodilatation in the lower extremities with substantial lowering of pressure and relief of symptoms. The procedure is mentioned because from this field or one closely related to it will likely come the solution of much of the troubles with hypertensive cases.

163. Hypotension.—*a.* Hypotension is that condition in which the systolic blood pressure consistently remains below 100 millimeters mercury. The condition is considered to be due to an atony of the cardiovascular system which is just opposite to the accepted theory of hypertension, spasm of the arterioles. Like hypertension, hypotension is divided into essential and organic types. Like essential hypertension, the cause of essential hypotension is unknown. Organic hypotension is the result of such conditions as congestive failure, coronary occlusion, surgical shock, hemorrhage, anemias, cachexia, alcoholism, cirrhosis, senility, and Addison's disease. It is likewise often seen as the terminal event in many of the acute infectious diseases. There is also to be seen a postural type in which the patient has paroxysms of hypotension upon assuming the upright position.

b. Essential hypotension is found in approximately 5 percent of the general population. Like hypertension, it runs in families and heredity is a very large factor. Geography and climate are factors and it is to be noted that in the south and in the Tropics hypotension is much more common, often being seen in approximately 10 percent of the population. Likewise, it is found in the asthenic types of individuals more than in the athletic or pyknic types. Well over half of all asthenics show a hypotension of some degree. In general, hypotensives seem to be slightly below par constitutionally, unable to indulge in sustained exertion, have low vital capacity, poor muscle tonus, and a generally poor physique. However, there are some very notable exceptions to this rule.

c. Symptoms are usually lacking in those cases whose systolic pressure remains above 90. Nervous instability is closely associated with the condition and most symptoms arise from that entity. However, transient attacks of weakness, giddiness, and dizziness which are

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probably due to transient spells of cerebral anemia do occur. When the systolic pressure gets below 90, these symptoms are accentuated; vertigo, syncope, and marked weakness setting in. Death is imminent in pressures below 80, although on several occasions systolic pressures as low as 50 have been recorded and recovery attained.

d. Signs are conspicously absent except the blood pressure readings. Most of the hypotensive individuals have a very youthful appearance and their apparent age is very deceptive. As the lower and more pathologic pressure levels are reached, the signs accompanying low pressures are seen, weak rapid pulse, distant heart sounds of poor quality, dilatation murmurs, pale colorless skin and membranes, subnormal temperature.

e. The prognosis in hypotension is very good for life. Insurance statistics show that persons with hypotension have an expectancy greater than average by approximately 35 percent. However, the life of an average hypotensive is a rather ineffectual one, full of neuroses, instability, and an incompetent type of physical activity. Hypotension has no effect upon the heart whatsoever, and produces no cardiac disease or end results other than those changes noted with shock and terminal low blood pressures.

f. Treatment is likewise rather ineffective. Foci of infection should be removed as an empirical procedure. Thyroid administration should be tried. Ephedrine sulphate may prove useful if it can be tolerated over long periods of administration. Adrenalin is useful in hypotensive emergencies. Cold baths, massage, and exercise are probably the most constructive measures to be used over long periods of time. These procedures, consistently indulged in, may maintain the systolic pressure at useful levels over long periods of time.

g. The Army has practically no place for hypotensives. Persons having systolic pressures persistently below 105 millimeters mercury are not acceptable for flying training. This measure is the result of a survey made in 1934 of hypotensive cases accepted for flying training. It was found that 90 percent of persons whose systolic pressures were below that level failed to qualify as pilots. The explanation is more or less obvious in view of the statements throughout the entire discussion of this condition.

164. Recommended texts.

Hypertension and Nephritis (Fishberg)	Lea and Febiger.
Heart Disease (White)	Macmillan.
Clinical Heart Disease (Levine)	Saunders
Diseases of the Heart (Lewis)	Macmillan.

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SECTION V

COR PULMONE

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165. General.—Cor pulmone is the cardiac end result of chronic pulmonary hypertension. Pulmonary hypertension, in turn, is caused by any of a number of factors, which increases the resistance encountered by the pulmonary circulation. Chronic pulmonary diseases, such as emphysema, chronic inflammatory conditions, and collapse of lung tissue, are the chief causes. In fact, emphysema is such a prominent factor in this disease that the condition is often called "emphysema heart." Cor pulmone is manifested chiefly by failure of the right side of the heart. In general, it can be compared to systemic hypertension and its resultant effect on the left heart.

166. Etiology.—*a.* The condition is rather rare, and has been reported in various series at less than 1 percent of all heart disease. It is a condition seen chiefly in older individuals, especially those over fifty. This age incidence is accounted for by the age incidence of the causative diseases. The other etiological factors are also those of the causative conditions.

b. As noted above, the causative conditions are chiefly the chronic pulmonary diseases. In addition to the commonest ones above-mentioned, endarteritis of the pulmonary vessels, pulmonary arteriosclerosis, diffuse pulmonary fibrosis, chest deformities, chronic pressure on air passages, and the industrial diseases, such as anthracosis, are given as causes. Pulmonary endarteritis (Ayerza's Disease) occurs in younger individuals than those mentioned above and is especially notable for its deep cyanosis. The depth of this cyanosis is often so marked that the individuals are called "black cardiacs." Massive pulmonary embolism involving over 60 percent of the lung tissue has been given recently as a cause of an acute type of cor pulmone. It is to be noted that failure of the left heart also raises interpulmonary pressure and results in failure of the musculature of the right side.

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167. Pathology.—The pathology to be noted is principally that of the underlying cause. The pulmonary artery is dilated and often shows atheromatous changes. The right ventricle itself at first hypertrophies and then, as failure sets in, dilates. Further than this initial hypertrophy of the right ventricle (and auricle subsequently) and the pulmonary artery changes, there is nothing to be noted until congestive failure of the right sided type sets in.

168. Symptoms.—There are no symptoms indicative of the condition. Those seen are the manifestations of the underlying pulmonary condition or those of the ultimate congestive failure of the right side of the heart. The dyspnoea and cough of chronic pulmonary disease are often mistaken for those of congestive failure, but familiarity with the failure symptoms of the two sides of the heart will make it easier to avoid this pitfall.

169. Signs.—Prior to congestive failure, the signs are those of the underlying condition. After congestive failure sets in, the signs of failure of the right ventricle are added. These are increased venous pressure, edema, ascites, anasarca, and passive congestion of the various abdominal organs. The right ventricular enlargement will be detected only by X-ray. The electrocardiogram shows the pattern of right ventricular strain, right axis deviation, elevation of RT_1 with compensatory depression of RT_{2-3}, and inversion of T_{2-3}.

170. Prognosis.—The prognosis is also that of the underlying pulmonary condition. The process in general is long drawn out, slow in its course, and causes varying degrees of cardiac invalidism over long periods of time. Congestive failure, when it does occur, merely adds to the seriousness of the general outlook. Death is usually due to the pulmonary condition rather than to the congestive failure.

171. Treatment.—The treatment of cor pulmone is essentially that of the underlying condition. When congestive failure sets in, the therapy is directed at its relief. The dominant principle of arranging a cardiac regime so that the patient is enabled to live within the limits of his cardiac reserve applies to this pulmonary-cardiac combination, as well as to the other cardiac conditions.

172. Massive pulmonary embolism.—The condition "acute cor pulmone," which occurs as a result of massive pulmonary embolism, is very rare and is seen chiefly in old people who have some underlying peripheral condition which will give rise to emboli. When 60 percent of the lung tissue is involved in embolism, acute dilatation of the pulmonary artery and the right side of the heart occurs. The symptoms and signs are those of the embolism, plus evidences of acute right ventricular failure, engorgement of the veins, pulmonary murmur,

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rapid thready pulse, and the lowered systolic blood pressure due to shock. The electrocardiograph shows a pulmonary embolism pattern, deep wide S_1, depressed RT_2, elevated RT_3, prominent Q_3, inverted T_3 and upright T_4. The prognosis at best is poor, although recovery has been reported. Treatment is essentially supportive, plus that of shock.

173. Recommended texts.

Heart Disease (White)	Macmillan.
Heart Failure (Fishberg)	Lea and Febiger.
Diseases of the Heart (Lewis)	Macmillan.

Section VI

CHRONIC NONVALVULAR HEART DISEASE

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174. General.—Chronic nonvalvular heart disease is that cardiac end result of senile degeneration which in the past has been labeled "chronic myocarditis." The term "chronic myocarditis" has fallen into disrepute because of the inference of inflammation that it carries and such other terms as "myocardosis," "interstitial myofibrosis," "myocardial fibrosis," and "chronic nonvalvular heart disease" have been substituted by various writers. Regardless of the name chosen, the condition is one of a progressive myocardial fibrosis; a progressive loss of cardiac reserve associated with advancing senility, often without a demonstrable pathologic basis, and which is characterized by symptoms and clinical signs out of proportion to the pathology shown at autopsy. It is a disintegration of the cardiovascular system, varied in its manifestations, which parallels the general physical disintegration of old age. In short, it is a wearing out process.

175. Etiology.—Except for age, the etiological considerations are varied and vague. The condition is seen in those past middle life and the older the age group studied, the higher the incidence. It is the cardiac accompaniment of the lowering vitality, failing nutrition, and wasting of tissues that characterizes the aging process. It is commonly associated with generalized arteriosclerosis, coronary sclerosis,

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syphilis, and hypertension. It is frequently found in connection with diseases affecting the general nutrition, such as diabetes, leukemia, and the severe anemias. Acute fevers, acute myocarditis, foci of infection, excessive use of alcohol (especially malt liquors), excessive use of tobacco, overeating, and a life of extreme physical exertion are considered to be contributing factors. All of these conditions are thought to hasten the progress of the myocardial fibrosis and contribute to its earlier appearance and greater severity.

176. Pathology.—*a.* There are no pathological findings definitely indicative of this condition. Often there are none at all demonstrable, even after a long and severe clinical course. Frequently the muscle will appear to be powerful, strikingly well nourished, and normal in color. It is a fairly conservative estimate to state that this lack of findings will exist in almost one-half of the cases.

b. When pathology is found, it is chiefly a wasting of muscle tissue with fibrous replacement. This wasting of muscle tissue occurs chiefly in the wasting diseases, cachexias, starvation, and tuberculosis. Here the muscle becomes pale; there is decrease in the size of individual muscle fibers; there is degeneration within the fibers as evidenced by deposits of hemosiderin and brown granules; and there is a reduction in the amount of fat tissue. A coincident increase in fibrous tissue occurs, which is both a deposit of fibrin between muscle bundles and a replacement of atrophic muscle fibers themselves. This atrophy and replacement process is not seen as frequently as the simple interstitial type of fibrosis. Here, the normal supporting connective tissue of the myocardium hypertrophies in varying degrees and embarrasses myocardial efficiency thereby. There is practically no atrophy of the myocardial fibers themselves, the process being merely an increase in the amount of supporting tissue. This is sometimes called cardiosclerosis. Along with this, an associated sclerosis of the coronary arterioles is frequently found. In fact, sclerosis of the coronary arteries and arterioles will be found in well over half of cases seen. In this nonwasting type, there is often also an increase in the amount of fat deposited about the heart, especially in the sulci. Sometimes this fat deposit is so large as to embarrass heart action in itself, especially when fat granules become deposited between muscle bundles. Frequently symptoms of mild cardiac insufficiency will be caused by this excessive deposit of fat alone, and the symptoms and signs will clear up remarkably upon simple reduction of weight. The process is called fatty infiltration, and it frequently exists in obese patients in the absence of cardiosclerosis. Myocardosis can also be produced, although somewhat rarely, by the fatty

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degeneration of muscle cells, which is reputed to accompany some of the acute infections and which does accompany certain chemical toxins. This very rare fatty degeneration is to be sharply differentiated from the common fatty infiltration described above, which is a normal accompaniment of obesity.

c. As the cardiac reserve diminishes, dilatation occurs. This dilatation further complicates the picture, in that the myocardium stretches to some degree, thereby further reducing its efficiency. The valve rings also stretch and become incompetent, producing the characteristic to-and-fro murmur of dilatation. Any valve defects here are purely secondary to dilatation and are not to be confused with the organic valvular conditions which also produce cardiac insufficiency and failure. This dilatation is the first step toward congestive failure and heralds the advent of the long train of signs and symptoms of that condition.

177. Symptoms.—Chronic nonvalvular heart disease is an entity which has no definite symptom complex associated with it. There is nothing specific which can be described as characteristic, unless a gradual loss of myocardial reserve of indefinite causation can be taken as such. The patient notices a progressive enfeeblement, progressive weakness, lowered vitality, and an increasing tendency to fatigue, both mental and physical. He notes that he is less and less able to carry on his routine functions and that he must curtail his activities more and more. Headache, irritability, vertigo, depression, and even mental deterioration are common. Insomnia is often bitterly complained of. The patient is frequently conscious of his heart action, both at rest and upon exertion, this consciousness often increasing in degree to an annoying palpitation or precordial ache upon smaller and smaller amounts of activity. A slight edema of the ankles is often present in the evening, noticed as a tightness of shoes or a bulging at the shoe tops. This disappears with rest, and is usually gone by morning. Increasingly frequent nocturia (which relieves this edema) further disturbs rest. Many patients exhibit a distinct dislike to lying on their left sides, merely stating that it is uncomfortable and that they seem to breathe easier in other positions. These symptoms of decreasing myocardial efficiency, plus those more pronounced symptoms of congestive failure, constitute the rather vague picture of cardiovascular-renal insufficiency which accompanies the progressive troubles of senility.

178. Signs.—The signs are those of a weakening myocardium. The pulse diminishes in force, volume, and strength. The heart sounds become diminished in intensity and quality, muffled, distant,

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faint. The loss of myocardial strength causes a decrease in the muscle component of the first sound until practically only the valvular component is left, thus equalizing the first and second sounds and creating a tic-tac quality. Gallop rhythm may be seen later in the course of the condition, due to either prolonged conduction time or to bundle branch block. This gallop rhythm may be due mechanically either to an additional third sound or a reduplication of the second. Pulsus alternans is seen in the terminal stages of the condition and is to be taken as an indication that the heart is struggling to perform the work of which it is barely capable. The signs of varying degrees of congestive failure or other complicating conditions may also be superimposed. The picture may be far from a simple one, in that all of the varying ailments of senility may be present as complications. X-ray is of very little help. The electrocardiogram presents a varied picture, ranging from apparent normality through amplitude decreases, slurring, notching, T wave changes, to blocks and arrhythmias. The electrocardiogram, in conjunction with symptoms and signs, is often very helpful and may be used to follow the progress of the case.

179. Prognosis.—*a.* The prognosis as to length of life is variable, each case being a law unto itself. The condition progresses insidiously on to death and the progress may hardly be detected by persons in constant attendance. Two very definite factors in evaluating prognosis exist, the amount of remaining cardiac reserve (to be estimated by exercise tolerance) and the amount of strain to which the heart is ordinarily or extraordinarily put. The latter is more or less controllable by a cardiac regime. Such factors as illness, intercurrent infection, extreme emotional upset, short periods of marked exertion and mental strain are definitely adverse in their effects. Death usually occurs during congestive failure, although a certain number of individuals will die peacefully in their sleep, sitting in a chair, or in other comparable circumstances. Those who die of congestive failure usually die quickly and exhibit very little response to treatment. All are more or less cardiac cripples for varying periods prior to death.

b. Two definitely bad prognostic signs exist, bundle branch block and pulsus alternans. Almost all cases in which a diagnosis of bundle branch block can be made electrocardiographically will be dead within 2 years. Almost all of those showing pulsus alternans will have died within a year; a very few will live as long as 2 years.

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180. Treatment.—*a.* Treatment consists essentially of rest and limitation of activity. Patients should avoid excesses, strain, exposure, and infection. Good hygiene is very helpful, and attention should be given to the details of securing satisfactory elimination and kidney action. Water intake should be governed by the capability of the cardiorenal system to handle it. Diet should be moderate and directed at reducing overweight or maintaining nutrition of those who are underweight. Proper amounts of rest should be secured if possible and mild sedatives should be given to secure a good night's sleep at intervals. Exercise is helpful if tolerated well, but special care should be taken to insure that such exercise is well within the limits of the cardiac reserve.

b. Medication should be entirely symptomatic and supportive. Tonic doses of digitalis are very effective in some cases and will often prolong the onset of congestive failure. These patients as a rule do not respond to tonics but often react adversely. Measures directed at increasing digestive efficiency, such as bile salts, dilute hydrochloric acid, or pepsin, frequently add much to the comfort of the patient. Sedatives should be given in all cases where indicated, especially in those showing irritability and insomnia. Aminophyllin may or may not be helpful, but should be tried in all cases where at all indicated. Theoretically the coronary dilator action of this drug should increase the flow of blood to the cardiac muscle in all cases where the coronary arteries are not too severely involved by sclerosis. At best, treatment in this condition is merely palliative and inconsistencies in results, the vagaries of which are more the rule than the exception, should be expected.

181. Recommended texts.

Diseases of the Heart (Lewis)---------- Macmillan.
Clinical Heart Disease (Levine)---------- Saunders.

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CHAPTER 5

END RESULTS OF CARDIAC DISEASE

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SECTION I

CONGESTIVE FAILURE

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182. General.—Congestive failure is that type of circulatory failure in which the heart is no longer able to maintain adequately the circulation of blood throughout the body. Here there is a venous stasis in which the vessels are overfull due to that inability. In the other type of circulatory failure (shock or peripheral failure), the peripheral vessels are relatively empty due to causes other than cardiac. The congestive type of failure is usually a terminal phase of cardiac diseases and is the most common mode of death seen in those conditions.

183. Etiology.—*a.* The causes of congestive failure can be grouped under one heading, that is, those conditions which cause a long continued extra burden to be imposed upon the heart. Thus the diseases which place the greatest strain upon the cardiac economy are the commonest causes of congestive failure. The most common of these are valvular defects, chronic hypertension, and coronary disease. The less common causes are congenital heart disease, constrictive pericarditis, thyrotoxicosis, chronic pulmonary disease (especially emphysema), and the rapid arrhythmias of long continued duration (auricular fibrillation, auricular flutter, and infrequently, paroxysmal tachycardia). A closer analysis will disclose the fact that the damage done by thyrotoxicosis is accomplished also by virtue of its long continued tachycardia. Acute conditions such as a fulminating rheumatic pancarditis, acute bacterial endocarditis, subacute bacterial endocarditis, acute glomerular nephritis, and pulmonary embolism will occasionally cause a congestive failure. In the majority of cases, how-

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ever, congestive failure is the culmination of long continued cardiac disease and the acute conditions listed above are rather unusual in the causative role.

b. The various types of heart conditions place the bulk of their extra burden upon a single chamber, a fact which will assume some importance when the mechanism of congestive failure is considered. Strain of the left ventricle and initial failure of that chamber are produced especially by aortic valvular disease (especially regurgitation), hypertension, coronary occlusion, and mitral regurgitation. Strain of the right ventricle and initial failure of that chamber are produced by mitral stenosis, pulmonary disease, and congenital pulmonary stenosis. However, the greatest cause of failure of the right ventricle is an already existent failure of the left ventricle. Strain upon both ventricles and practically simultaneous failure of both chambers are produced by the long-continued rapid rates, thyrotoxicosis, arteriovenous fistula, general coronary involvement, and diffuse myocardial disease.

c. Although congestive failure can occur at any age, most failures will be seen in the older age groups. It is most common after the age of 50, over 70 percent of failures occurring after that age. The sex distribution, geographical occurrence, hereditary and environmental considerations are necessarily those of the underlying cause. Congestive failure is the most frequent accompaniment of heart disease; well over half of all the cases of organic cardiovascular disease will undergo the changes of venous stasis.

184. Pathology.—*a.* There is no pathology, either gross or microscopic, which is characteristic of the condition other than the passive congestion of the various tissues affected. Practically all of the soft tissues of the body are subject to the accumulation of fluid due to the venous stasis, and with but one or two exceptions, all of the symptoms and signs arise from this passive congestion. The pathological lesions characteristic of the underlying structural abnormality which was the primary cause of the cardiac insufficiency are seen chiefly in the heart. There is so little seen microscopically or grossly in the myocardium itself which portrays myocardial failure that a pathologist can hardly state from examination of the heart alone, whether or not failure existed. Therefore, nothing further will be said on this subject, other than to point out that cardiac dilatation, and often hypertrophy, are almost universal accompaniments of congestive failure.

b. An understanding of the mechanism of congestive failure is based entirely on the concept that one or more of the chambers of the heart is unable to meet adequately the work load placed upon it. That inability to meet this demand means dilatation, ineffective activity of

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the muscle, and an incapacity of the chamber to empty itself satisfactorily with each contraction. This inability to empty itself means that a slightly less than normal amount of blood is propelled forward, that the residuum in the chamber furnishes resistance to blood entering the cavity. This, plus the stretching of the valve rings due to the dilatation and the backflow through the relatively insufficient valves, add considerably to the work of the cardiac chamber immediately antecedent. Soon this strain on the antecedent chamber causes its failure and the process is ultimately carried backward to the great veins. More specifically, initial failure of the left ventricle adds considerably to the burden of the left auricle, which likewise ultimately fails, becomes engorged, and produces back pressure in the pulmonary veins. This increase in pulmonary venous pressure causes a pulmonary stasis, which results in an increase in general pulmonary pressure and adds considerably to the burden of the right ventricle. As a result of its increased burden, the right ventricle fails, then the right auricle, and pressure then mounts in the peripheral veins, causing venous stasis.

c. Initial failure of the right ventricle causes back pressure into the right auricle and on into the peripheral venous tree. Here the right ventricle is unable to pump the required amount of blood through the lungs into the left heart and both the systemic and coronary circulations suffer an inadequacy. The coronary inadequacy, especially, leads to myocardial difficulties which, in the course of time, involve the left ventricle in failure. The process as herein described is usually one of very slow progression, and the failure of each unit in its succession is a relatively long drawn out affair. Further, the process may be broken at any point by rest or digitalis, providing that the muscle itself possesses sufficient reserve to stage a come-back . Usually the initial failure of either ventricle is present in a minimal state over a period of years, very slowly progressing to an evident failure. The evident failure is often precipitated by some trivial extracardiac event, such as intercurrent infections, overexerton, overeating, overexcitement, or emotional upset. More commonly, however, cardiac failure is of very gradual onset, the evidences of congestive failure arising so insidiously that they are disregarded by the patient until they are of such moment as to demand some measure of correction. Further, it should be stated in all fairness that the above-given mechanism of cardiac failure is not universally accepted and the student is again referred to the standard texts for details of other theories.

185. Symptoms.—*a.* By far the most important symptom of congestive failure is dyspnoea. In fact, dyspnoea is so important that the examiner can almost date the onset of congestive failure by the initial appearance of breathlessness, and also gage the degree of fail-

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ure by the amount of exertion required to produce that breathlessness. Early in the condition shortness of breath is minimal and appears only upon moderate exertion. The individual first notices a slight increase in breathing upon undertaking activities which formerly caused no appreciable distress. Later, as the degree of failure progresses, the dyspnoea increases and the patient notes that lesser and lesser amounts of activity are necessary for him to become out of breath. Now he notes that climbing stairs, walking up an incline, or running for a bus causes marked distress. As the failure further progresses, he becomes breathless upon ordinary activity, and finally dyspneic at rest. The terminal stage of the condition can be said to be present if the individual has difficulty breathing when at rest even in the upright position. The cause of breathlessness is passive congestion of the lungs. As the congestion increases, the severity of the symptoms caused thereby also increases. Fairly early in the disease patients will note that breathlessness is increased by the recumbent position, that one or two pillows under the head give relief. As the condition grows worse, more and more pillows are required until the patient gets relief only in the upright position. Subjectively, the more advanced stages of dyspnoea cause the patient greater degrees of discomfort than any of the other symptoms. A sense of suffocation is most commonly described by patients as being by far the most distressing of all the features of congestive failure.

b. Other symptoms of lesser importance are weakness, fatigue, precordial ache, edema, ascites, and those occasioned by passive congestion of the gastrointestinal tract, lungs, and central nervous system. Weakness and fatigue are noted early in the course of the condition, and advance progressively, roughly paralleling the degree of failure. Weakness and its counterpart, easily induced fatigue, are the result of the arterial ischemia which, in turn, is due to the diminished output of the left ventricle. The precordial discomfort is more a continuous ache of mild to moderate character, than definite cardiac pain. In fact, cardiac pain which previously existed usually disappears with the onset of obvious congestive failure. Palpitation is rare in the absence of arrhythmias. Edema and ascites, while not symptoms in themselves, very frequently cause discomfort of varying degrees. In the earlier stages, passive congestion of the gastrointestinal tract causes indigestion, flatulence, and dyspepsia. Later, the passive congestion of the lining mucosa of the stomach creates a marked gastritis with nausea, vomiting, epigastric discomfort, and inability to take nourishment. The liver becomes engorged, enlarged, causing hypogastric discomfort, tenderness and, upon oc-

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casion, moderately severe pain accompanied by a sense of hypogastric fullness and heaviness. Disorders of liver function add to the dyspepsia. Rarely there is a resultant jaundice. Passive congestion of the lungs causes, in addition to the breathlessness noted above, cough with the production of frothy and ofttimes blood-tinged sputum. Pleural effusion of the right-sided unilateral type is characteristic of cardiac failure and adds its degree of thoracic discomfort. Passive congestion of the central nervous system leads to insomnia, headache, nervousness, mental disturbances, such as illusions and hallucinations, and often frank psychoses. Somnolence, delirium, and coma are common. Earlier in the course of failure, symptoms similar to those of cerebral arteriosclerosis are noted: irritability, amnesias, inability to concentrate, forgetfulness, weakness, faintness, dizziness, and occasionally, epileptiform seizures. It is to be emphasized that the above-given symptoms are, in general, evident in a more or less bilateral type of failure. Failure of the left ventricle alone gives symptoms almost completely limited to the lungs: breathlessness, congestion, and effusion. The occurrence of paroxysmal dyspnoea is thought to be pathognomonic of left ventricular failure. Cheyne-Stokes type of respiration is an occurrence seen in the more advanced stages. Failure of the right ventricle alone gives symptoms of peripheral venous stasis: edema, ascites, anasarca, gastrointestinal tract and central nervous system manifestations. The symptoms characteristic of each type of failure complement each other as the failure progresses, and by the time failure is complete practically all of them will be present.

186. Signs.—*a.* Congestive failure presents three signs of major importance, edema, cyanosis, and dyspnoea. All are due to venous stasis. Edema is the earliest and most easily detected and the one which dominates all other findings. It is of the pitting type, midway in degree of fluidity between the solid edema of myxedema and the fluid edema of nephrosis. It is characterized by a tendency to collect in the dependent portions of the patient's anatomy, in the feet and ankles of the ambulent patients, and in the buttocks, hips, thighs, and genitalia of those in the reclining position. It is rarely seen in the head, face, neck, upper chest, or hands. It varies in amount from small accumulations noted in the evening as a swelling over the shoe tops, which disappears after a night's rest, to the marked swelling of dependent portions, ascites, hydrothorax, and anasarca. In cardiac disease, effusion is seen earlier and in greater amounts in the right pleural cavity, whereas in most pulmonary types of effusion, the amount is practically equal on both sides. Edema, or more correctly termed "passive congestion," also

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occurs in brain, gastrointestinal tract, liver, spleen, kidneys, and lungs. Cyanosis also varies widely in degree from a faint "highness" in color of the lips and mucous membranes to purple blue of the severe degrees. It is to be noted especially in the mucous membranes, sclera, lips, tongue, and fingernails. Dyspnoea has already been discussed as a sympton, but the very obviousness of the air hunger and the contortions of the patient in attempting to aerate adequately the lungs makes it an obvious sign. The degree of apparent dyspnoea will vary directly with the degree of myocardial failure.

b. There are several other related signs of congestive failure, all of lesser importance, and most of them due to passive congestion of the viscera. First, the general appearance of the patient is one of malnutrition if the condition has existed any length of time and there is usually an associated, more apparent than real, anemia. Second, there is pulsation of the jugular veins when the patient is in the upright position. When the heart is functioning normally, jugular veins do not pulsate when the neck is above the level of the heart, and any pulsation of these veins (or any others) when above the level of the heart is an infallible sign of increased venous pressure or venous stasis. Third, the lungs present coarse, moist rales, heard first at the bases, later as the degree of failure progresses, at progressively higher levels. In addition, the vital capacity of the lungs diminishes in direct proportion to the reduction of available air space caused by the passive congestion of that organ. Fourth, the heart presents signs of enlargement, an element of which is practically always dilatation. The sounds are usually of poor quality, often assuming the tic-tac characteristic. The murmurs of dilatation are present, often superimposed on those of the underlying organic cause. Gallop rhythm sometimes occurs and when present usually indicates a serious myocardial status. Fifth, the liver becomes engorged, enlarged, and tender. It can often be felt far below the costal margin. Infrequently, the engorgement of the liver produces a jaundice which further complicates the skin color, or, if the engorgement persists over a long enough period of time, cirrhosis develops. Sixth, the passive congestion of the kidneys causes definite urinary changes. The urine output is scanty, concentrated, shows albumin, hyaline casts, phosphates, and often red blood cells. When the edematous fluid begins to empty itself under the influence of rest or digitalis, the picture reverses itself and the urine becomes voluminous, of low concentration, and presents very little solid matter. Seventh, the pulse is usually of poor quality, rapid, and often arrhythmias are present. Auricular fibrillation and premature ventricular beats are

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the most common of the arrhythmias. Eighth, blood pressure is usually maintained at a much lower level than that shown prior to failure. In cases of hypertension, the systolic pressure often falls markedly, but the diastolic pressure remains fixed. The fall in blood pressure is gradual and parallels the severity of the congestive failure in general. Ninth, in the more advanced cases, fluid collects in all of the serous cavities, peritoneum, pleurae, and pericardium. While this phenomenon is rightly a part of the advanced stages of edema, it is mentioned here again for emphasis. X-ray is of little or no help, and usually shows nothing but enlargement, which as has been noted, can either be hypertrophy, dilatation, or both. The electrocardiograph, likewise, offers little assistance other than denoting arrhythmias, blocks, coronary or hypertensive findings. There is nothing definite in any of the electrocardiograph patterns which is indicative of congestive failure.

c. In delineating the signs of congestive failure, the student is further cautioned to bear in mind the fact that they will vary characteristically with the type of ventricular failure. In primary failure of the left ventricle, the pulmonary signs will predominate, whereas in primary failure of the right ventricle, the systemic signs will be most marked. In those cases where the strain is bilateral, or the progress of the failure advanced to the point of bilateral involvement, practically all of the signs listed will be present in varying degrees.

187. Prognosis.—*a.* The prognosis of congestive failure varies widely with the cause, severity, and the response to treatment; so much so that it is almost axiomatic that no attempt should ever be made to predict the length of life. Congestive failure which is the result of cardiovascular lues, aortic valvular lesions, or coronary occlusion is to be considered serious, no matter how slight the degree of that failure. The average outlook in these cases is but a few months to a few years after the onset of failure. Congestive failure which is the result of hypertension or mitral stenosis is not to be considered in so serious a light, in that these cases can often be carried along for years in a satisfactory state of compensation by the use of a cardiac regime and digitalis. Congestive failure which is due to hyperthyroidism or the controllable rapid rates carries a very good outlook, in fact an absolute cure without fear of recurrence can be promised in many instances in which hyperthyoidism can be corrected by a subtotal thyroidectomy. In the matter of severity it quite naturally follows that the more severe the failure the more serious the outlook, but this certainty must be tempered by consideration of cause as outlined above. In considering severity the damage done by congestion of the various systems must also be taken into account. Often edema

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produces leg ulcers which become infected and very resistant to treatment, or a renal insufficiency may develop or cerebral edema may cause an exhausing delirium. In the matter of response to treatment, the factor of myocardial reserve is dominant. The nearer normal the heart is prior to the onset of failure (as in the case of failure produced by some unavoidable excessive strain in the presence of a moderately damaged heart), the more cardiac reserve remains with which to stage a come-back under rest and digitalis. The reverse is true in the aged where the heart has gradually lost its reserve with the advancing years, and in the presence of congestive failure, there is no capacity within the muscle itself to stage that come-back.

b. Several points in prognosis are worth noting. In general, the average duration of life after the onset of congestive failure is but a few years. The first attack of congestive failure is rarely fatal. Persons who have done manual labor all of their lives have a better outlook than those who have done desk work or inactive indoor occupations. A congestive failure which had its onset while the patient is on a regime of rest is of very serious outlook.

c. Death in congestive failure is due to cardiac exhaustion, intercurrent infection, or to the overwhelming severity of one of the systemic manifestations such as uremia or delirium.

188. Treatment.—*a.* The treatment of congestive failure varies with the degree of failure present, and every case must be considered as an individual problem, the therapy being adjusted within broad general limits to fit each situation. For the purposes of convenience the description of the treatment of congestive failure will be divided into two groups, that of the mild to moderate failure and that of the acute, disabling type. Treatment of both groups is based on the same principles, although in the latter severer type it is necessarily more extensive and complicated.

b. In the mild to moderate degrees of failure, all of the symptoms and signs are minimal or little more than minimal, and the patent is usually able to carry on to some degree his usual activities. Here restriction of activity, plus a varying amount of extra rest, may be all that is necessary. The patient should be taught the value of moderation in all things: activity, exercise, eating, smoking; and should especially be taught to care for himself in such a manner as to avoid contracting any type of infection. Here also much constructive good can be done by treatment of the causative condition when that condition is amenable to treatment as is hyperthroidism and, to some extent, lues. Frequently, however, the failure is too far advanced to respond adequately to the simple measures outlined above and some degree of medication must be instituted. Here digi-

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talis is almost a specific, and inasmuch as the indications are usually not urgent, the patient should be digitalized by the slow method. Once digitalization has been accomplished, it should be maintained as long as needed, and withdrawn only when sure that the failure has receded to such a point that the regime of rest and moderation will maintain the patient in a state of compensation. Other medication in mild to moderate degrees of failure is essentially symptomatic.

c. Treatment of the acute, severe, disabling stage of congestive failure is much more drastic and much more involved. The procedure can be more readily discussed under five headings, given in the order of their importance: rest, medication, psychology, diet and fluid intake, and symptomatic relief.

(1) Rest is the most important single procedure and it accomplishes its purpose by reducing the amount of work the heart is called upon to do. Rest should be absolute, both mental and physical, insofar as it is possible to attain that end. The patient should be in bed with the head elevated by means of pillows and so supported by pillows or other means that he will expend no effort in maintaining that position. An adjustable hospital bed is ideal for this purpose. Occasionally a case will be seen in which the dyspnoea is so severe that the maximum comfort is obtained by sitting in a chair. This position should not be discouraged, and the patient should be made as comfortable as possible with the thought kept in mind of returning him to his bed as soon as symptoms allow. Good nursing care is essential to complete rest, with alcohol rubs, padding for joints, and all of the details which attain that end. Later, as the patient improves, the rest regime need not be so absolute and may be relaxed in proportion to the improvement of the patient.

(2) Medication is easily second in importance to rest and discussion can be limited to three general groups—digitalis, sedatives, and diuretics.

(*a*) Digitalis has two main indications, the relief of congestive failure and the slowing of abnormally rapid cardiac rates. In congestive failure, it slows the pulse and increases the efficiency of the myocardium, enabling the heart to empty itself more completely with each beat. It thus aids in the removal of the excess fluids of edema, abolishes cyanosis, breathlessness, and the disturbing symptoms of congestion of the gastrointestinal tract and the central nervous system. When digitalis is used, it should be used to full effect or until the appearance of toxic symptoms, whichever is first. Full effect is considered to be the slowing of the pulse, establishment of polyuria, and the relief of dyspnoea. Toxic symptoms (early) are

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nausea, vomiting, diarrhoea, blurring of vision, drowsiness, mental confusion, and coupled beats. Two methods of attaining full effect exist, rapid and slow. Both aim at the attainment of full digitalization. The rapid method is the one of choice in acute disabling congestive failure and the slow method is the one of choice in the less urgent cases described earlier under treatment. The rapid method consists of the administration of 0.2 to 0.3 gram of the powdered leaf or its equivalent every 6 hours to full effect. Twenty-four to forty-eight hours are required for digitalization. The slow method of digitalization consists of the administration of 0.1 gram of the powdered leaf or its equivalent t. i. d. to effect. A week to 10 days is required for digitalization. After full digitalization has been attained, it must be maintained. Maintenance of effect is simply the replacement of digitalis excreted by the body. This excretion is cared for by the administration of from 0.1 to 0.2 gram of the powdered leaf daily. The exact dosage varies with each case and must be judged individually for each person. Thus, in the severe congestive failures, complete digitalization must be attained by the rapid method and that digitalization maintained until the cardiac failure is abated, or often indefinitely when it is not completely abated. The use of digitalis substitutes (thevetin, ouabain, etc.) is not recommended at any time as it is believed that any cardiac emergency can be handled with the intravenous preparations of digitalis now available. For further information on this procedure, the student is again referred to the larger texts.

(*b*) Sedatives should be used generously throughout the course of acute congestive failure. Morphine is the drug of choice in the severer stages and should be used unsparingly. The advantage of morphine here is that it allays fear, induces sleep, slows down the bodily activity and metabolism, reduces discomfort and, in general, aids immeasurably in the attainment of rest when it is so badly needed. Further, morphine will often relieve the subjective aspects of dyspnoea to a marked degree and thus further add to the general comfort. Later, as the degree of failure diminishes, the milder sedatives should be substituted in doses which should parallel the improvement. Phenobarbital in rather large doses is the drug of choice to substitute for morphine in reducing activity, discomfort, and fear. Sodium amytal should be used to induce sleep when needed. Bromides and chloral hydrate are also useful and should be used freely either as a substitute for the barbiturates or in alternation with them.

(*c*) Diuretics should be used freely in the presence of edema and effusions, and their use continued until both have disappeared. There are two types to choose from, the purines and the mercurials. Both

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have their firm advocates. The purines are slower acting than the mercurials and much less effective. Theocin, theophyllin-ethylenediamine (aminophyllin), theobdomin-sodii-salicylate (diuretin), and theobromin-calcium-salicylate (theocalcin) constitute the most usable of the purine group. For methods of use and dosage the student is referred to the literature. Of the mercurials, salyrgan is more or less typical, and can be used intravenously or intramuscularly, never subcutaneously. The dose is 1 cubic centimeter of the 10 percent solution per day, or on alternate days, as long as needed. It is well to begin with half a cubic centimeter for the initial dose as a test for susceptibility. The drug is very effective and huge quantities of edematous fluid may be excreted in one day as a result of its use (10,000 cc. in a case of nephrosis). The action of salyrgan is enhanced by the simultaneous administration of ammonium chloride in 60 to 120 grain doses daily. Its action is even more enhanced if time is available to give this drug for 3 days prior to the administration of the mercurial. The best of the other types of diuretics is intravenous hypertonic glucose. This accomplishes diuresis in cases where, for any reason, other diuretics are contraindicated, or when intravenous nourishment is desired. It is best used during shock or in cases of congestive failure due to diphtheria. The dose is 25 to 50 grams of glucose in 20 to 50 percent solution intravenously per day. Much benefit is derived from its use in the so-called "dry failures."

(3) The value of psychology in the treatment of any illness hardly needs comment. In congestive failure it is of the utmost importance. Patients may be bolstered up by judicious handling, their fears markedly allayed by the establishment of confidence, their whole structure of hope built up by a few well chosen remarks as to their outlook or favorable progress. The fear of impending death is very prominent, and those who lose hope seem to do very poorly. On the other hand, obvious misrepresentation of the truth, will destroy confidence very quickly. However, emphasizing even the smallest signs of improvement and minimizing the adverse factors are helpful. Specifically, in ward work, place a new congestive failure next to one who has done well, or better yet, between two who are convalescent. Daily comment is directed at the signs of improvement, the slowing of the pulse, the easier breathing, and the amount of edema fluid passed. The fluid and dietary restrictions are removed very obviously, reduction in medication commented upon and pointed to as signs of improvement. In short, everything possible is done by ac-

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tion, word, and attitude to convey to the patient the hopefulness, rather than the hopelessness, of the condition.

(4) Restriction of diet and fluids during the acute stage of the condition accomplishes a double purpose, easing the load on the digestive tract, and thus indirectly that of the heart, and by decreasing somewhat the amount of fluid with which the cardiovascular system has to cope. The diet of choice during the first 3 or 4 days is the Karrell diet, 200 cubic centimeters of milk four times daily. Whiskey in 15- to 30-cubic centimeter amounts can often be given advantageously between milk feedings, and combining this with an equal amount of cream is often very helpful to those used to large amounts of food. Chipped ice to suck adequately relieves the thirst occasioned by this regime and the individual rarely gets more than 100 to 200 cubic centimeters extra fluid by this method in 24 hours. No other fluids or salt should be given during this period. After 2 or 3 days, or when the edema begins to move, this strict regime can be relaxed somewhat and a salt-poor maintenance diet can be instituted. Total fluid intake should be limited to 1,500 cubic centimeters during this period and gradually increased as the patient's condition warrants. The increase in fluids can be made much of from the psychological point of view and each item added to the diet can be stressed as a sign of improvement. Favorite items of food can be added when they conform at all to the diet, and such items often add much to the patient's happiness and comfort.

(5) Symptomatic relief, although the least important in the grouping, should not be at all neglected. Here the comfort of the patient may be materially benefited and often the return of compensation may be hastened. First and foremost under this heading should be listed the mechanical removal of excess fluids. Paracentesis for the removal of ascitic, pleural, or pericardial fluid gives a very marked degree of relief and will sometimes prove to be the turning point in a case which seemed otherwise hopeless. Tapping of excess fluid is highly recommended in those cases in which the response to digitalis and diuretics is slow or in those cases where either drug is contraindicated. Caution should be exercised as to the amount of fluid removed each day in that too sudden removal of a large quantity of transudate may upset the cardiovascular equilibrium and prove fatal. The use of Southey tubes or their equivalent (Wassermann needles and Dakin tubing) is recommended for use in cases where peripheral edema is not moving satisfactorily. In pulmonary edema, venesection of from 300 to 500 cubic centimeters of blood will often prove lifesaving. Marked and unresponsive dyspnoea calls for relief by the administra-

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tion of oxygen either by tent, mask, or catheter. The use of stimulants may often tide an individual over a crisis. Caffeine-sodium-benzoate in 7½-grain doses intravenously is an old reliable procedure. Even black coffee orally will accomplish the same purpose if the urgency is not too great. The value of adrenalin in 5 to 10 minim doses of $\frac{1}{1000}$ solution intramuscularly is well known. The commercial preparation coramine has proved effective on several occasions. Considerable cardiac distress arises from the gastrointestinal tract and may be relieved with peppermint water or chloroform water. There are many other procedures of value which could be included, but only one will be mentioned. Subtotal thyroidectomy after proper preparation will completely relieve congestive failure when that congestive failure is due to hyperthyroidism. Some authors in the recent past have recommended this procedure in all cases of congestive failure and even in angina pectoris, but in the light of present knowledge, caution is advised before using this procedure in the absence of hyperthyroidism.

d. After the patient has shown enough improvement to be removed from the acute, critical classification, and compensation is returning, a very definitely planned convalescence must be followed. There should be a gradual reduction in all medication, such reduction to be made as rapidly as the patient's condition warrants. Even digitalis should be dispensed with if the patient's heart is able to carry its burden without the help of the drug. Diet and fluid intake should be increased gradually as the improvement justifies, with the aim in view of restoring both to as near normal as possible. The patient should gradually leave the bed, sitting up for longer periods each day, preferably in a wheel chair, so that he can enjoy the advantages of such changes of scenery as are available. As soon as he is able to walk, he should be encouraged to do so within the restrictions of fatigue, increasing the period of exertion daily within the limits of the returning cardiac reserve. Graded exercises are helpful in the later stages of convalescence. It should be reemphasized that arteriosclerotics and the elderly do poorly when kept too long in bed, and bearing this thought in mind, special effort should be made to restore them to activity at a point much earlier than would seem warranted in a younger individual. Occupational therapy of various natures is extremely useful in helping to keep the patient's mind and hands occupied, and will markedly assist in maintaining morale and in making the days seem to pass more quickly while waiting for the heart to regain its adequacy. The convalescence will vary widely in length and results with the cardiac reserve and the amount of cooperation

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received from the patient, but in general the better planned the convalescence, other factors being equal, the better the outlook for the patient in the immediate future. In short, much future trouble can often be avoided by the judicious management of convalescence.

189. Recommended texts.

Heart Failure (Fishberg)	Lea and Febiger.
Failure of the Circulation (Harrison)	Williams and Wilkins.
Diseases of the Heart (Lewis)	Macmillan.
Heart Disease (White)	Macmillan.
Clinical Heart Disease (Levine)	Saunders.

Section II

SERIOUS ARRHYTHMIAS

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190. General.—The more serious cardiac arrhythmias which are discussed below arise chiefly in damaged hearts as a result of the disease which caused the damage. Very occasionally paroxysms of these arrhythmias may occur in the absence of any demonstrable heart damage, but this is the exception rather than the rule. These more serious conditions are seen principally in the older individuals. Those individuals below forty in whom attacks occur are the victims of the advanced valvular diseases or of a precocious myocardial involvement. All of the arrhythmias discussed in this section are serious and carry a definitely bad prognostic outlook. Therefore, an individual subject to them should not be on flying status as long as they exist.

191. Auricular flutter.—*a.* Auricular flutter occurs in two forms, paroxysmal and permanent. It is not at all common and occurs roughly 5 percent as often as auricular fibrillation. The permanent form is especially rare, the condition usually being paroxysmal and a transitional stage between the normal or the less serious auricular rhythms and auricular fibrillation.

b. The focus of stimulus formation arises in the auricular muscle outside of the sino-auricular node. The most plausible explanation of this mechanism seems to be that these impulses arise from a single irritable point in the auricular muscle and differ in this respect from auricular extrasystoles and paroxysmal tachycardia only in the rate

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of impulse formation. The more widely accepted theory is that these impulses arise as a result of a "circus" movement around one of the great veins. The rate of auricular contractions varies from 150 to 400 per minute and is usually too rapid for a ventricular response to each beat. Hence a physiological block occurs, giving rise to a slower ventricular rate which bears a direct mathematical ratio to the auricular rate, 2 to 1, 3 to 1, 4 to 1, etc. Thus, the ventricular rate is relatively slow and may vary from 50 to 150 per minute. The rhythm is regular except when the ratio is changing.

c. Flutter comes and goes abruptly, thus resembling a simple paroxysm of tachycardia. Flutter, however, is more persistent than tachycardia, and although it normally lasts but a few hours or days, may last even for weeks or months. In this respect it resembles auriclar fibrillation. Flutter may pass spontaneously into fibrillation, but more commonly the change is induced by digitalis. This fact is used in the treatment of the condition. Other than the duration of the attacks, flutter differs from paroxysmal tachycardia only in the presence of a physiological block. A 1 to 1 flutter cannot be distinguished from paroxysmal tachycardia.

d. The actual causation of flutter is unknown. It is associated prominently with such diseases as mitral stenosis, arteriosclerosis, coronary sclerosis, coronary thrombosis, thyroid heart disease, and hypertension. It is occasionally seen in the acute infections during childhood. In a small percentage of cases it is entirely idiopathic. The attacks themselves are often induced by strain, exertion, excitement, and many illnesses, but just as often occur spontaneously. Although no characteristic pathological changes have been found, the presence of the condition indicates some degree of myocardial damage.

e. The symptoms vary from those associated with short paroxysms of tachycardia to those of congestive failure. The burden thrown upon the heart is proportionate to the increase of ventricular rate. The degree of ventricular muscle insufficiency depends chiefly on the rapidity of the ventricular contractions and the state of the myocardium. When the heart muscle is degenerate and the rate rapid, dilatation, engorgement of the veins, enlargement of the liver, and anasarca appear. On the other hand, when the condition develops in a heart muscle which has considerable reserve, signs of failure are not manifested. There is, however, always a limited response to effort, and palpitation is common. Occasionally the ventricle assumes the full auricular rate and distressing symptoms, especially loss of consciousness, occur. Such attacks must of necessity be short, otherwise death would occur. Unfortunately flutter is extremely difficult to

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diagnose clinically. The electrocardiograph is usually necessary for its diagnosis. Nevertheless, there are a number of associated signs which are suggestive of flutter. A regular, persistent ventricular rate of over 120 to 160 per minute at rest and not increased by effort in an elderly subject should suggest flutter. Short paroxysms of flutter are to be differentiated from paroxysmal tachycardia by the fact that in the latter the ventricular rate is usually from 180 to 220 per minute. The ventricular rate in flutter seldom exceeds 150 per minute. Again, attacks of paroxysmal tachycardia seldom last as long as attacks of flutter. If a rapid ventricular rate of 150 persists for a month, the condition is almost certainly flutter. Vagal pressure is more apt to produce slowing of ventricular action in flutter than in paroxysmal tachycardia. A rapid ventricular rate that is suddenly halved or doubled is almost certainly flutter.

f. Auricular flutter with irregular ventricular action due to varying degrees of block can be differentiated from auricular fibrillation by a simple exercise test. Exercise in flutter immediately accelerates the ventricular rate and produces a regular pulse, whereas in fibrillation the irregularity is increased. In flutter, when the ventricular action is regular, exercise often will have no effect on the rate. Flutter with a 3 to 1 or 4 to 1 heart block, which produces a ventricular rate within normal limits, is practically impossible to diagnose by ordinary clinical means. Occasionally, in such cases, inspection of the jugular veins will show the rapid regular impulses transmitted from the fluttering auricles.

g. The prognosis in general is unfavorable. Auricular flutter is associated with myocardial degeneration. It impairs the efficiency of the heart, but is not in itself a dangerous condition providing the heart muscle is not extensively diseased. The ultimate prognosis is based on the degree of burden produced by the rapid ventricular action and the evidence of the extent of myocardial degeneration. The effect of treatment is of value in determining the prognosis in individual cases.

h. Digitalis is of greatest value in the treatment of flutter as it always reduces the ventricular rate by increasing the block between the auricles and ventricles. Having obtained a reduction in the ventricular rate, if digitalis then is given in large doses, flutter is often converted into fibrillation. If digitalis is then withdrawn, fibrillation often ceases and the normal rhythm is resumed. This may persist for years. The treatment of auricular flutter by digitalis often brings with it great improvement in the patient's general con-

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dition. As the normal rhythm replaces the rapid ventricular rate, the signs of cardiac failure disappear rapidly.

192. Auricular fibrillation.—*a.* Fibrillation of the auricles is a condition in which coordinate contractions of the auricles are replaced by fibrillary twitchings. Here the sino-auricular node has lost its function as the pacemaker, which function is taken up by a multitude of individual foci in the auricular muscle. Each of the new foci give out stimuli at a rapid rate, the result being that only a small local area of auricular muscle reacts to each. Only an occasional one of these fibrillary contractions is strong enough to stimulate the auriculoventricular node, hence the irregularity of the latter's response. The average rate of the auricle's fibrillation discernible on the electrocardiograph is 450 per minute.

b. Normally, the auricles contract in response to a stimulus which originates in the sino-auricular node, and this contraction is a coordinated movement which drives blood into the ventricles. This coordinated activity ceases when fibrillation begins. The auricles become virtually paralyzed, dilate, and stand in tremulous diastole. The ventricles no longer receive the normal stimuli from the auricles, but instead receive numerous and haphazard impulses. As a result the ventricular rate is raised and the contractions occur in a totally irregular manner.

c. The number of stimuli that succeed in reaching and stimulating the ventricles is governed mainly by the conductivity of the bundle of His. This junctional tissue usually puts some check on the number passing through it. When the conductivity of the bundle is good, the ventricular rate reaches 100 or more. Digitalis diminishes this conductivity and thus by increasing the block, slows the rate of the ventricles. This fact is utilized to good advantage in treatment of the condition.

d. The rapid and irregular action of the ventricles produces a totally irregular pulse. This has frequently been described as "irregular irregularity." The force, volume, and rhythm (spacing) of each beat is different from the others. There is also a marked discrepancy between the number of ventricular contractions and the number of pulse waves which are felt at the wrist. Many ventricular systoles, particularly when the ventricular rate is rapid, either fail to open the aortic semilunar valves or expel a sufficient quantity of blood to cause a palpable arterial pulse. This discrepancy between the number of ventricular systoles and the number of pulse waves felt at the wrist is termed pulse deficit.

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e. The causes of auricular fibrillation are essentially those listed under auricular flutter. The actual causation is unknown. Mitral stenosis, arteriosclerosis, coronary thrombosis, thyroid heart disease, hypertension, acute infections, malignant endocarditis, congenital heart disease, and luetic involvement are the diseases listed. Less frequently than with auricular flutter, no causative condition can be shown. The attacks themselves are often induced by strain, exertion, excitement, many illnesses, or overindulgence in alcohol or tobacco.

f. The pathological anatomy is uncertain. From the intimate relationship that exists between auricular premature contractions, auricular tachycardia, and auricular fibrillation, it seems probable that all are produced by similar pathological changes. Apparently, some cases are in reality examples of disordered function with no underlying structural change.

g. Fibrillation is in most instances a permanent irregularity and, when present, signifies cardiac muscle damage. Occasionally, however, patients are observed who show transient attacks of fibrillation. These begin and end suddenly and may last for a few hours, days, or weeks. This condition is termed paroxysmal auricular fibrillation. In such cases, the heart function may not be seriously impaired. Fibrillation is often recurrent, the paroxysms being separated by weeks, months, or years of normal heart rhythm. It seems probable that in many patients fibrillation first appears in the form of paroxysms, and that these later become permanent.

h. The condition may exist for long periods of time without causing symptoms. When symptoms are present they are usually limited to palpitation in its various phases. Here the patient is conscious of the irregular and forceful action of the heart. Often this is very annoying and distressing. Dyspnoea and precordial ache are sometimes also present in mild degrees. The element of fear accentuates all of the symptoms and often superimposes those of a cardiac neurosis. When congestive failure sets in, the symptoms of that condition are added. It is interesting to note that with the onset of fibrillation, in cases of angina pectoris, most cases lose their painful attacks for the duration of the arrhythmia.

i. Fibrillation of the auricles can generally be recognized clinically wthout graphic aid. In most cases palpation of the pulse and apex beat is sufficient. However, the electrocardiogram makes the diagnosis absolute. The pulse shows an absolute arrhythmia. The pauses between the beats are continually changing, and two successive beats are rarely of the same strength. The more rapid the pulse the more marked is the irregularity. The disorderly action of the

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ventricles is readily appreciated at the apex. The heart sounds are modified; they vary in intensity, now a rapid run of weak sounds, now a mixture of weak and strong sounds occur in totally irregular fashion. With a ventricular rate of 100 or more, there is usually a marked pulse deficit.

j. Fibrillation can usually be differentiated from heart block and premature contractions with marked ventricular irregularity, by the fact that moderate exercise increases the irregularity in fibrillation and diminishes or eradicates it in the other two conditions. Fibrillation, also, is usually a permanent condition, while premature beats are present only from time to time. A marked characteristic of fibrillation is the exaggerated response of the heart rate to exercise. The following simple test is of aid in the diagnosis. If the heart beats at a rate of 120 or over, or can be induced by any means (such as exercise) to beat at such rates, while the pulse remains irregular, fibrillation is almost certainly present. A heart rate which is unduly accelerated by effort, and which still remains irregular at the accelerated rate, is characteristic of auricular fibrillation. Irregular flutter, blocks, and extrasystoles are usually abolished by exercise.

k. Auricular fibrillation is most often a sign of some myocardial change—an evidence of myocardial damage. At times, it is the chief or only reliable sign that the heart muscle has been affected. Permanent auricular fibrillation is one of the most common causes of cardiac failure of the congestive type, and few patients survive its onset for more than 10 years. The ultimate prognosis depends on the efficiency of the heart muscle, that is, the ability of the heart to function under the new rate and rhythm. A persistent ventricular rate of 100 or over is usually a serious sign. On the other hand, single paroxysms occuring in the course of acute infections usually do not recur and are probably not indicative of permanent damage to the heart. A most valuable aid to prognosis may be found by observing the manner in which patients respond to treatment. If two patients showing equal signs of cardiac failure differ in that one presents a normal rhythm while the other manifests a rapid ventricular action, the result of fibrillation, then the prognosis is more favorable in the latter, for the latter is more amenable to treatment.

l. It is in this condition that digitalis has gained its reputation in the treatment of cardiac disease. In fibrillation, digitalis slows the rate of the ventricles by increasing the block between the auricles and ventricles. In fibrillation the upper end of the bundle of His is constantly bombarded by numerous stimuli from the fibrillating auricles, and the ventricular rhythm is determined by the number of

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these stimuli that reach the ventricles. When the conductivity of the bundle is depressed by digitalis, the ventricles beat more slowly and often more regularly, even though the abnormal auricular activity continues. With a slower and more regular ventricular action, each ventricular contraction becomes mechanically effective and the pulse deficit tends to disappear. The great benefit which follows the use of digitalis in such cases is due mainly to the slower ventricular rate, which gives the heart more rest. This, in turn, leads to an improvement in the contractile power of the heart, and thus causes a more efficient circulation.

m. In about 50 percent of cases, quinidin converts auricular fibrillation to regular sinus rhythms. However, the use of quinidin is distinctly dangerous and an elaborate set of precautions should be carried out in its use. The student is here referred to the larger texts for details of its administration.

193. Ventricular tachycardia.—Tachycardia of ventricular origin is uncommon. Recent cases have been reported in which poisoning by digitalis or quinidin has induced this type of paroxysm. The ventricular complexes in the electrocardiogram are distinctly aberrant (abnormal in shape) and resemble the complexes of ventricular premature contractions. In fact, the condition may be considered a rapid succession of ventricular premature contractions. Ventricular tachycardia is closely allied to ventricular fibrillation, as it has been shown that ventricular tachycardia may at any time pass over into ventricular fibrillation. When ventricular tachycardia is present, the prognosis is definitely bad, in that the finding of this condition always means serious myocardial damage.

194. Ventricular fibrillation.—*a.* This condition is an important cause of cardiac death, and probably is the unrecognized mode of death in many cases. The condition, when it does exist, is of short duration because ventricular fibrillation is incompatible with life and causes death to supervene in a very few moments. Recovery is rather rare.

b. The condition is known to occur with coronary thrombosis, electrocution, benzol poisoning, chloroform poisoning, and with angina pectoris. The mechanism of the condition is similar to that of ventricular premature contractions and ventricular tachycardia, portions of the ventricular muscle becoming irritable and producing impulses. In the case of ventricular fibrillation (as with auricular fibrillation), multiple foci arise, each sending forth its own impulse to stimulate a small neighboring area. The result is a cessation of coordinated ventricular contractions, with replacement by tremulous

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fibrillary type of muscle contraction, with which little or no blood is expelled into the aorta. It is practically impossible to diagnose clinically, and of little value because death or resumption of coordinated ventricular activity supervenes very shortly. There is no treatment of any proved value. Quinidin sulphate or acetyl-beta-methylcholin is reported to be of some value in the prophylaxis of the condition.

195. Recommend texts.

Heart Disease (White)---------- Macmillan.
The Mechanism and Graphic Registration of the Heart Beat (Lewis)---------- Shaw and Sons.
Clinical Heart Disease (Levine)---------- Saunders.

Section III

HEART BLOCK

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Sino-auricular block---------- 197
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196. General.—Heart block is the result of a depression of the function of conduction system tissues at some point along their course. It is usually seen in the older-age group as one of the end results of cardiac disease. Classification is made according to the points along the conduction system where the block can occur: sino-auricular, auricular, auriculoventricular, and intraventricular.

197. Sino-auricular block.—*a.* Sino-auricular block is that condition in which impulses fail to arise at the sino-auricular node, the dominant pacemaker of the heart. Sino-auricular block may be partial or complete. In partial block, there is an interval between two auricular beats which is equal, or almost equal, to the length of two normal beats. In complete block, there is a complete absence of impulses from the sino-auricular node for a period of a few beats. This condition is also called auricular standstill, due to the absence of auricular beating. Here, the auricuoventricular node assumes the function of pacemaker for the period of time involved. It is to be noted that some authorities consider sinus bradycardia as a type of sino-auricular block.

b. Sino-auricular block is due to sympathetic inhibition, or vagal stimulation, either of which may be produced by a variety of condi-

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tions. Pressure on the carotid sinus is notable for causing periods of complete sino-auricular block. Drugs such as digitalis or quinidin will sometimes accomplish the same effect. Certain acute illnesses, and the condition, jaundice, have the same result. Although sino-auricular block is relatively rare, it is seen most commonly in young individuals.

c. The condition causes few symptoms or signs. Partial block causes none. Complete block sometimes causes palpitation, weakness, and dizziness. Prolonged block causes syncope. About the only sign of note is the assumption of a very slow pulse rate for a period of time, due to the establishment of idioventricular rhythm. However, this is so apt to be missed that it is of little clinical value. Electrocardiographic tracings make the diagnosis absolute when those tracings can be secured during an attack.

d. The condition has little or no clinical significance. It is a transient occurrence that will be detected usually by accident. Treatment, when indicated, is atropine subcutaneously for attacks, ephedrine or adrenalin at appropriate intervals for control of repeated attacks.

198. Auricular block.—Auricular block is of no clinical importance. Very little is known of the condition, and it is practically unrecognizable even in electrocardiographic tracings. It has been included only for completeness of this classification.

199. Auriculoventricular block.—*a.* Auriculoventricular block is that condition in which there is a delay in the conduction of the impulse across the auriculoventricular node or the tissues of the bundle of His above the bifurcation. The condition has been divided for convenience of classification into three degrees of seriousness, named first, second, and third degrees. First degree block is simply a prolongation of the PR interval of the electrocardiogram greater than 0.20 second. Second degree block is simply a further slowing of conduction, in which ventricular beats are dropped occasionally or at regular intervals. Third degree block is a complete dissociation between auricles and ventricles, in which each pair of chambers beats at its own rate entirely independent of the other. Auriculoventricular block can be either temporary or permanent. Often it fluctuates from one grade to another, momentarily assuming a different degree. This fluctuation is especially prominent in the temporary type and here exercise, vagal stimulation, or digitalis will often convert a lower degree block into third degree. The causes of auriculoventricular heart block are numerous. The temporary type is usually caused by toxic conditions or substances such as diph-

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theria, rheumatic fever, influenza, pneumonia, uremia, digitalis, quinidin, or even asphyxia. Overstimulation of the vagus could be added as a functional cause. The permanent type is usually caused by such organic factors as degeneration, fibrosis, or ischemia, which are in turn, caused by coronary disease, senility, syphilis, the end results of rheumatic fever and diphtheria, or by pressure on the node and bundle resulting from endocarditic lesions, neoplasms, or trauma. Digitalis, rheumatic fever, diphtheria, and the coronary diseases are by far the most common causes. Temporary blocks are usually less in degree and much more common than permanent blocks, which are usually of higher degree and much less frequent. Blocks occur more commonly in the older age groups, in fact fully four-fifths of all blocks will be seen in individuals past fifty. The older the individual in whom a block occurs, the more apt that block is to be organic and of a high degree of severity. Another peculiarity of auriculoventricular block is that when the condition exists during the very rapid rates, such as auricular flutter or fibrillation, it is a beneficial process cutting down the work of the ventricles tremendously; but when it occurs at the slow rates, it is definitely deterimental.

b. The pathology of auriculoventricular block is rather uncertain. It varies from the finding of no lesions (even in some high grade blocks) to the finding of marked fibrosis in or around the node and bundle. Either inflammation, degeneration, or ischemia precedes the fibrosis, although any or all may coexist in the same heart. Also, any of the four may be found in or around the conducting tissue without the functional manifestation of block. Often massive infarctions occur which miss the bundles entirely, whereas small rheumatic, luetic, or endocarditic processes may localize in the bundle and cause a block of high degree. At best, the pathology of this condition is rather puzzling.

c. First degree block is merely the prolongation of the PR interval of the electrocardiogram above the upper limit of normal duration, which is 0.20 second. This is entirely an electrocardiographic diagnosis, there being no clinical findings indicative of the condition. The three main causes are digitalis overdosage, acute rheumatic fever, and diphtheria. The occurrence of first degree block in any of these conditions is evidence that the heart is becoming seriously involved.

d. Second degree block is also known as incomplete or partial heart block. It has two subdivisions, that of occasional dropped beats and that of regularly dropped beats. In the former, conduction across the auriculoventricular node gradually slows down until an occasional auricular impulse finds the ventricle in the refractive state, and hence

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no ventricular response occurs. In the latter subdivision this conduction is slowed down to such a degree that the ventricle fails to respond to every sixth, fifth, fourth, third, or second impulse, and the ventricular beat is missed. Thus a 6 to 5, 5 to 4, 4 to 3, 3 to 2, or 2 to 1 rhythm is established, and the pulse shows rhythmic irregularity. This regular irregularity is fairly easily diagnosed with practice, and is to be compared with the previously mentioned irregular irregularity of auricular fibrillation. Heart block of this degree creates no symptoms other than occasional palpitation. All other symptoms that are seen are due to the underlying causes. Likewise, all signs except the dropped beats and the electrocardiographic findings are due to the underlying causes. Although this condition can be diagnosed clinically with but slight acumen, the electrocardiograph makes the diagnosis absolutely and simply.

e. Third degree block is also known as complete heart block. This condition occurs when the second degree block becomes greater than 2 to 1 and the heart rate thus becomes less than 30 to 35 per minute, or when the delay becomes so great as to prevent any supranodal impulses from reaching the ventricle. When this occurs, the auriculoventricular node takes up its own rhythm independently of the sino-auricular node. (The AV node also assumes its own rhythm when the normal impulses are slow in reaching the node, when they are formed at a slow rate, or when an unusually long interval occurs between them.) Here the rate is less than 40 per minute, usually being between 30 and 35. The rhythm is usually regular. Infrequently, impulse formation at the auriculoventricular node becomes irregular, unusually slow (8 to 20), or stops for a period of from 1 to 2 beats up to 20 to 30 seconds. Here symptoms more serious than the palpitation seen in second degree block occur. The omission of four or five beats will cause transient weakness, dizziness, or faintness. A ventricular standstill of 10 to 30 seconds duration, or a very slow rate, will cause Adams-Stokes syndrome. This syndrome is described as "attacks of unconsciousness, often accompanied by muscular twitchings or even general convulsions, occurring in auriculoventricular block when the ventricular diastole is sufficiently prolonged to result in a severe grade of cerebral ischemia." * These attacks also occur during a shift from partial to complete block, before the ventricles can establish the new rhythm. Temporary cessation of heart action causing the syndrome may occur at widely separated intervals or may

*New York Health and Tuberculosis Association "Criteria for the Classification and Diagnosis of Heart Disease."

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occur with marked frequency. Cessation of heart activity for much more than a minute results in death. The sign of complete heart block is the very slow pulse. Any pulse rate below forty is more than apt to be a complete block. (Rarely it may be sinus bradycardia, or even more rarely yet, nodal rhythm.) A compensatory rise in systolic blood pressure produces a moderately widened pulse pressure. All other signs except those of Adams-Stokes syndrome or the lesser degrees of ventricular standstill are those of the underlying condition. Here, as in all other blocks, the ultimate diagnosis is made by the electrocardiogram.

f. The prognosis of auriculoventricular heart block is good in the absence of acute disease or old age. If due to drugs, simple removal usually suffices to accomplish relief. Idiopathic heart block seen in youth is often compatible with long life. In acute disease or old age, the finding of block adds to the seriousness of the prognosis because of the implication that the heart is seriously involved. Specifically, people with persistent heart block of any degree usually die within a few years. Death is usually from the underlying condition or congestive failure. Rather rarely ventricular standstill is a cause of death. In this possibility, ventricular standstill, lies most of the dangers arising from complete heart block.

g. The treatment of auriculoventricular block is usually that of the underlying condition, and the establishment of a cardiac regime under which the individual confines his activities within the limits of his heart's capacity. In certain acute conditions, such as diphtheria and often in syphilis, specific treatment accomplishes remarkable results. When drugs or toxins (digitalis, quinidin, etc.) are the cause, simple removal is effective. Adrenalin subcutaneously is useful in Adams-Stokes syndrome when the equipment and drug are available during an attack. Ephedrine is effective for prolonged usage in the prevention of these attacks. Barium chloride, atropine, and thyroid extract have also been tried and found helpful in certain cases. In some of the more persistent cases it may be necessary to give all of the drugs a trial in the hope of finding one which is valuable in the individual ease at hand.

200. Intraventricular block.—*a.* Intraventricular block is that condition in which the conduction of impulses over one or the other of the branches of the bundle of His is defective. A more common name is bundle branch block. Like first degree block, it is entirely an electrocardiographic diagnosis. This condition is often associated with other types of block, especially the auriculoventricular type. Like auriculoventricular block, the condition may be either

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temporary or permanent. Causes, frequency, and age incidence also parallel the auriculoventricular type with one exception, that coronary sclerosis by far outweighs all other conditions in causitive importance. The condition may be complete or incomplete, the differentiation between the two being of major importance in considering prognosis. Pathology is essentially ischemia, degeneration, and fibrosis. The pathology otherwise is indefinite as in auriculoventricular block, and like that condition, the correlation between functional bundle branch block and pathological findings is equally as puzzling.

b. Symptoms other than those of the underlying condition are absent. Likewise, signs other than those of the underlying condition are absent, with the following exceptions: splitting of the heart sounds, or gallop rhythm, are frequent accompaniments, and in their presence bundle branch block should be suspected. Pulsus alternans is another sign which when present is very frequently accompanied by intraventricular block. As in all of the other blocks, the electrocardiograph makes the absolute diagnosis.

c. Prognosis is always poorer in the presence of bundle branch block, and definitely bad when the condition is complete. A finding of intraventricular block always means myocardial pathology, the temporary type often of a fleeting toxic nature, the permanent type indicating accomplished organic damage. Incomplete bundle branch block is often compatible with a fairly long and active life and just as often an accompaniment of advancing degeneration. Complete bundle branch block indicates an average duration of life of a few years. Some authorities restrict this figure to 1 or 2 years following discovery of the lesion. Treatment is limited to that of the underlying condition and the establishment of a cardiac regime compatible with the functional capacity of the heart.

201. Recommended texts.

Heart Disease (White)	Macmillan.
The Mechanism and Graphic Registration of the Heart Beat (Lewis)	Shaw and Sons, Ltd.
Clinical Heart Disease (Levine)	Saunders.
Clinical Disorders of the Heart Beat (Lewis)	Shaw and Sons, Ltd.

SECTION IV

SUDDEN DEATH

	Paragraph
General	202
Recommended texts	203

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202. General.—*a.* Death from cardiac disease usually occurs as a result of congestive failure, myocardial exhaustion, or some intercurrent infection. However, in a small percentage of cases, death is unexpectedly sudden and dramatic. The immediate cause of this type of death is one of five mechanisms; ventricular fibrillation, ventricular standstill, ventricular rupture, aortic rupture, or intracardiac thrombus. Ventricular fibrillation is seen in coronary disease, both insufficiency and occlusion; poisoning by chloroform, benzol, quinidin, digitalis, and probably other toxins; and in electrocution. Ventricular standstill is due either to heart block or to reflex vagal action which results in paralysis of the pacemakers. This reflex vagal stimulation is seen in angina pectoris and coronary occlusion. Ventricular rupture is the result of weakening of the heart wall by infarction or rarely by bacterial endocarditis. Aortic rupture is the end result of aneurysm and aortitis. Rather rarely a ball valve thrombus will form within one of the chambers of the heart completely blocking one of the valve orifices, or a large embolus will block the aorta or the pulmonary artery. The former is seen especially in mitral disease, the latter in systemic or pulmonary venous thrombosis.

b. In any of these instances death occurs within a few seconds to 1 or 2 minutes. Sudden heart deaths are all of this type. Fairly rapid deaths, accompanied by a few minutes to several hours of coma or shock, are not directly due to any of the causes given above. This slower type of death is seen in the cerebral accidents, peripheral emboli, or the major visceral accidents and is frequently confused with the cardiac type of death.

c. Treatment is limited entirely to those cases of ventricular fibrillation or standstill due to toxic causes or electricity which occur under more or less controlled circumstances. Intracardiac injection of adrenalin is recommended by some authorities in cases of reflex standstill or even in fibrillation, but it is doubtful if worthwhile results are often accomplished. Others recommend massage of the heart (in operative cases where the abdomen is open, slapping the chest, or pricking the heart with a needle. In the recent past, stimulation of the heart by an electric current carried through a double needle has been tried with but indifferent success.

203. Recommended texts.

Disease of the Heart (Lewis)---------- Macmillan.
Heart Disease (White)---------- Macmillan.
Clinical Heart Disease (Levine)---------- Saunders.

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CHAPTER 6

EFFECTS OF ALTITUDE UPON HEART

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General ---- 204
Effects of prolonged residence ---- 205
Changes occurring upon ascent ---- 206
Other factors ---- 207
Problem of flight with an abnormal heart ---- 208
Who shall fly ---- 209
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204. General.—In considering the problem of the effects of altitude upon the heart, concern is chiefly with its effect on short stays such as ordinary flying rather than with the effects seen upon prolonged residence at high altitudes. However, inasmuch as an understanding of this latter subject lends clarity to the former, it will be discussed briefly. Further, with the recent development of the long-range bombers, flights as long as 48 hours become a distinct possibility. Therefore, what was formerly a fairly simple problem involving chiefly the changes occurring from oxygen deprivation with short ascents, now becomes complicated by additional factors, such as the cumulative effects of fatigue and the cumulative effects of prolonged partial anoxemia. However, the basic problem involving cardiac patients still involves the effects of oxygen deprivation of the type seen in ordinary flying with its fairly rapid ascents and descents.

205. Effects of prolonged residence.—The effects of prolonged residence in altitudes above 12,000 feet can be very aptly labeled as those of chronic anoxemia. The problem is one of acclimatization to the lowered oxygen tension. The principal changes taking place are an increase in both the size and number of red blood corpuscles, an increase in hemoglobin content, an increase in the size and capacity of the chest, a tachypnoea, and an increase in the size of the heart, especially of the right side. This increase in heart size very much resembles cor pulmone and its effects. All of the changes listed above are seen to be an effort of the system to get more of the rarified air into the lungs and a normal amount of oxygen to the tissues. Individuals who live at high altitudes, no matter how well acclimated, are physically more inefficient than those living at lower levels. The same amount of work causes much greater degrees of effort syndrome in mountaineers than it does in normal individuals living at sea leval.

206. Changes occurring upon ascent.—*a.* In discussing changes occurring upon ascent we must differentiate at once between gradual

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and rapid ascents. Gradual ascent is that slow, laborious type used by mountain climbers and expeditions attempting to reach great altitudes in which definite periods of acclimitization at various levels are allowed. Rapid ascent for our purposes is considered to be the ordinary rate of climb of the average airplane, rather than the sudden lowering of oxygen tensions so often used by laboratory experimenters. In the prolonged flights of bombers, mentioned previously as a possibility, both types must be taken into consideration to a certain extent.

b. The changes occurring in a gradual ascent are essentially those seen in normal individuals during short stays at high altitudes or upon first reaching high altitudes. Here there is increased respiratory effort immediately. The amount of that effort expended varies directly with the altitude reached. If the stay at higher levels is prolonged, this effort decreases as acclimatization occurs, although some degree of tachypnoea will always persist. After a few hours' stay, the resting pulse accelerates from 25 to 75 percent. This acceleration varies somewhat with the altitude attained, although it is not affected to the degree that the dyspnoea is. After 24 to 48 hours' stay, the pulse gradually begins deceleration, to its previous normal, which point will be reached in from 1 to 2 weeks. The pulse response to eating, excitement, and exertion is in proportion to the new resting rate. The blood picture changes after 24 hours' stay. The red blood cells and the hemoglobin increase rapidly for the next 5 or 6 days, and then continue that increase at a slower rate until adequate compensatory changes have taken place. The blood pressure lowers somewhat, but not markedly, and its response to exercise, exertion, or excitement is much exaggerated during the early weeks or months of the stay at high levels. The venous pressure becomes slightly lowered and remains lowered throughout the stay.

c. Rapid ascents to high altitudes present a somewhat different problem. Here anoxemia is the dominant factor and it manifests itself both psychologically and physically. Normally no changes will be noted at altitudes below 10,000 feet. Between 10,000 and 15,000 feet dyspnoea becomes of some consequence. Above 15,000 feet the dyspnoea becomes acute but is overshadowed by the psychological breakdown which is manifested by decreased attention, distractibility, faulty judgment, and less often by such responses as euphoria, anger, fear, destructiveness. Physically, the sensations are dulled, vision is restricted to a narrow field, hearing is impaired, coordination is

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poor, voluntary muscles are poorly controlled, and drowsiness and sleepiness ensue. Above 22,000 feet unconsciousness supervenes.

d. The increased depth of respiration is the most constant factor of all of the changes noted. This varies directly with the altitude reached, but like all of the factors noted above is subject to variation from individual to individual. The respiratory rate increases but little in normal individuals prior to the fainting level. The pulse rate increases somewhat, but not markedly. The greatest increase occurs only after a few hours. Sinus arrhythmia becomes prominent, probably due to the increased depth of respiration. The blood pressure changes are inconstant. Usually in normal individuals the systolic pressure falls slightly. Prior to fainting the systolic pressure rises, then falls, the diastolic pressure falls and the pulse rate usually increases. Van Liere reports that there is an acute cardiac dilatation (animal experimentation) as a result of anoxemia, the degree of which varies with the degree of the anoxemia. The electrocardiographic changes upon anoxemia are essentially a lowering or inversion of the T waves, a depression of the R-T interval, and sometimes a deformity of the QRS group. The dilatation and the EKG changes are considered to be the direct result of anoxemia of the myocardium itself. In regard to the efficiency of the heart at high altitudes, at 15,000 feet the heart works 20 percent harder to accomplish 20 percent less work. This rule apparently holds proportionately true at lower and higher altitudes.

e. Use of oxygen enables man to make rapid ascents above 15,000 feet without the effects noted above. However, even with oxygen, the absolute ceiling for man (using the open method of administration) is near 43,000 feet and the actual attainable ceiling by this method is somewhat below that. Therefore, so long as short flights are kept below a critical level of 15,000 feet, or if above that level oxygen is used, the effects noted will be minimal.

207. Other factors.—On longer flights, as with the new long range bombers, the problem becomes more complicated. Not only must the changes taking place on rapid ascents and the changes discussed as occurring during the early hours of residence at high altitudes be considered but also the effects of fatigue. Ordinary flying produces fatigue far out of proportion to the effort expended, and far greater than an equal period of hard work at lower levels would produce. While this fatigue is largely mental, emotional, and neuromuscular, it also affects the neurocirculatory system to some degree, this probably

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being due to the increased load on the heart occasioned by altitudes. There is a definite increase in the minute volume index on prolonged flights, which increase indicates an increased load on the heart. The cumulative effects of prolonged partial anoxemia encountered on prolonged flights at lower altitudes are also a factor in increasing the burden on the system in general and quite logically also on the neurocirculatory system. The exact nature and degree of these cumulative effects have not yet been determined. The compensatory blood changes which would take place on a prolonged flight can be ignored insofar as the heart is concerned. Further, there are other factors, the effects of which have not yet been completely worked out. These are: the effect of velocity, the effect of centrifugal or centripetal force, the effect of reduced atmospheric pressure other than the anoxemia induced, the effect of rapid changes in atmospheric pressure, and the effect of rapid thermal changes.

208. Problem of flight with an abnormal heart.—It can be seen from the above that flying does definitely affect the normal heart. This effect is undoubtedly exaggerated when a diseased heart is involved. We know from experiments that blood pressure changes in hypertensives subjected to high altitudes are much more marked; that hypertensives are more subject to the fainting reaction; that the fainting reaction in all cardiacs is more easily induced than in normal individuals; and that cardiacs are not only subject to increased depth of respiration but that their respiratory rate increases, sometimes markedly, as it does not do in normals. Experiments have shown that attacks of angina pectoris can be induced by placing the individuals subject to the attacks in lowered oxygen tensions. The effect on persons with coronary disease can easily be imagined. Likewise, the effect of myocardial anoxemia induced by altitudes on weakened cardiac musculature, such as is seen in most of the advanced stages of organic heart disease, must be appreciable. The vasomotor collapse and the acute dilatation seen in the higher altitudes is definitely damaging. The effect of partial anoxemia on borderline congestive failures is easily imagined. Therefore, it is evident in the light of the above facts and of clinical experience with diseased hearts under other types of strain, that flying is definitely detrimental and dangerous in heart disease. The higher and the longer the flight, the more work the heart is called upon to do and the more cumulative fatigue and anoxemia impose themselves upon the cardiovascular system. It is evident, therefore, that persons with valvular disease of any moment whatsoever, those with syphilitic, arteriosclerotic, or

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hypertensive involvement or with senile degeneration should not be allowed flying duty. In these cases, the additional burden of the repeatedly minor insults to the cardiovascular system occasioned even by short flights can conceivably hasten or precipitate marginal failure. The same holds true for persons with coronary disease or angina pectoris, because they are more subject to anginal attacks or even thrombosis. The added burden of flying in these cases may precipitate these latter events and, as well as damage the heart, may end disastrously. In short, the additional strain which flying places upon the diseased heart adds materially to its burden, lessens the flying efficiency of the individual, creates a poorer response to altitude, and conceivably decreases the cardiac reserve with each insult, no matter how minimal.

209. Who shall fly.—The question of how much cardiac disease can be tolerated in duty pilots is one which must be settled individually in each case. One rule (purely arbitrary) is whether or not the individual can do twelve continuous hours of physical labor without an abnormal cardiovascular response, or without further damaging his heart. If so, he can pilot an airplane. Obviously this rule excludes all cardiac disease of any moment whatsoever. The question then arises, who shall be placed on observer status and who shall ride passively in airplanes as passengers. For the Army, this requirement (observer status) is rather severe, in that observers must also go to high altitudes and often remain there for long periods. For commercial passengers it is quite a different story, for here, except over the mountains, flights are relatively short and at lower altitudes. For observer status there should be very little relaxation of the rule above given for pilots. However, inasmuch as the risk is almost entirely personal (no other lives involved as with the pilot), and the training and experience of these individuals are valuable to the service, some latitude can be given. Here again, it is an individual problem. In general, the questions to be answered are: "Is he physically sound other than his heart? Is his cardiac damage minimal? Is there any suggestion of early congestive failure? Is he subject to anginal attacks?" If the first two can be answered "yes" and the other two "no", then he can fairly safely fly as an observer, even though he has detectable heart damage. As to commercial passengers, only the last two questions are applicable, and then there is some doubt as to whether the fourth one applies. The use of oxygen in airliners can conceivably carry over almost any cardiac who is able to get about fairly satisfactorily under his own power.

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210. Recommended texts.

Aviation Medicine (Armstrong 1939)	Williams and Wilkins.
Physiology Notes (Snell)	School of Aviation Medicine.
Heart Disease (White)	Macmillan.
Aviation Medicine (Bauer)	Williams and Wilkins.

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[A. G. 062.11 (8–26–40).]

BY ORDER OF THE SECRETARY OF WAR:

G. C. MARSHALL,
Chief of Staff.

OFFICIAL:

E. S. ADAMS,
Major General,
The Adjutant General.

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